VOLUME LXI MARCH, 1951 NUMBER 3

UNIVERSITY
OF MICHIGAN
APR 9 1951
MEDICAL
LIBRARY

THE LARYNGOSCOPE

FOUNDED IN 1896
BY
MAX A. GOLDSTEIN M.D.

PUBLISHED BY
THE LARYNGOSCOPE
640 SOUTH KINGSHIGHWAY ST. LOUIS (10), MO., U.S.A.

NOTICE TO CONTRIBUTORS

THE LARYNGOSCOPE reserves the right of exclusive publication of all articles submitted. This does not preclude their publication in Transactions of various Societies.

Manuscripts should be typewritten, double spaced, on one side of paper only and with sufficient margins to allow for corrections.

References should be complete: author's surname, initials, title of article, Journal, volume, page, month, year.

Six illustrations will be furnished for each article without cost to author. Authors will please limit illustrations to six or assume the expense of additional illustrations.

Proofs will be submitted to authors for corrections. If these are not returned, articles will be published as corrected in this office.

Reprints will be furnished at the following prices:

WITHOUT COVER

	250 Copies	500 Copies	1000 Copies	2000 Copies
Four Pages	$11.75	$13.75	$18.25	$28.25
Eight Pages	23.25	29.25	40.50	61.50
Twelve Pages	34.75	45.50	66.00	104.00
Sixteen Pages	45.50	56.00	76.00	115.50
Twenty Pages	54.00	67.00	92.50	143.00
Twenty-four Pages	65.00	80.50	110.50	169.00
Twenty-eight Pages	76.75	93.50	127.00	193.00
Thirty-two Pages	86.00	104.00	140.00	212.00

WITH COVER

Four Pages	$22.25	$27.75	$38.00	$60.50
Eight Pages	33.75	43.25	60.25	93.75
Twelve Pages	45.25	59.50	85.75	136.25
Sixteen Pages	56.00	70.00	95.75	147.75
Twenty Pages	64.50	81.00	112.25	175.25
Twenty-four Pages	75.50	94.50	130.25	201.25
Twenty-eight Pages	87.25	107.50	146.75	225.25
Thirty-two Pages	96.50	118.00	159.75	244.25

Express charges will be paid by consignee.

THE
LARYNGOSCOPE.

VOL. LXI MARCH, 1951. No. 3

NASOPHARYNGEAL CANCER.*†‡§

DANIEL MILLER, M.D.,
Boston, Mass.

PURPOSE.

From time to time a review of the literature and a re-evaluation of the management of specific disease entities seems necessary. In such a review some phase of the disease such as diagnosis, therapy, racial or geographic distribution is often specifically emphasized. Since one of the distressing features of our subject—nasopharyngeal cancer—is the time-lag in arriving at a proper diagnosis (as compared with early diagnosis of cancer in other parts of the body) a desire to reduce this delay has been the stimulating force for our review. It is apparent that early diagnosis in cancer of the nasopharynx will mean more cures in this previously considered hopeless disease.

Although cancer of the nasopharynx is strictly an otolaryngological disease by its primary location, accurate knowledge of its pathogenesis must be considered essential for those in the three major specialties of neurology, ophthalmology and otolaryngology, as well as for the general practitioner. It must

*Submitted as Candidate's Thesis to American Laryngological, Rhinological and Otological Society, Inc., January, 1951.

†Pondville Hospital (Massachusetts Department of Public Health). P. O. Walpole, Mass. Reprint number 143.

‡Massachusetts General Hospital, Boston, Mass.

§Massachusetts Eye and Ear Infirmary, Boston, Mass.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 5, 1951.

be emphasized that such knowledge is essential to the general practitioner since the patient for the most part sees his family physician first. Hauser and Brownell[1] stated it well by saying that "nasopharyngeal cancer should be suspected by the general practitioner and diagnosed by the otolaryngologist with ease."

It is alarming to read the literature and discover that so many patients with cancer of the nasopharynx had not had correct diagnoses made until it was too late for cure; and that, in addition, such patients had been subjected to innumerable and unnecessary operations in the belief that their symptoms were due to some other disorder. H. E. Martin and Blady[2] reported that 46 per cent of their patients had received surgical treatment to relieve symptoms before a correct diagnosis was established. Simmons and Ariel[3] reported that 12.7 per cent of their series had been operated upon for symptomatic relief before a correct diagnosis was made. This surgery included tonsillectomies, mastoidectomies, radical sinus operations, dental extractions and many other procedures. In 1931, New[4] stated that 185 operations on 194 patients with malignant lesions of the nasopharynx had been performed for symptomatic relief prior to admission to his clinic; among these were 56 nasal operations, several mastoidectomies, repeated myringotomies, alcohol injection for trifacial neuralgia, and numerous dental extractions. Kleinfeld,[5] in 1936, reported that 15 of his 40 cases had had 20 useless operations, done from one to six months before admission, ranging from craniotomy to radical sinus and mastoid surgery. Needles[6] reported 35 cases treated prior to 1937 with 12 patients having been subjected to unnecessary operations before correct diagnoses were made.

HISTORY.

Although cancer of the nasopharynx is not a very common disease, it is still sufficiently prevalent not to be considered among the diseases rarely encountered. As additional attention is given to this disease more cases will be uncovered. This seems evident in view of the marked increase in the number of cases reported since its first description.

In 1924, Smith and Dawson[7] reported finding erosion of the base of the skull in a Byzantine mummy and they thought it might be due to a nasopharyngeal cancer.

In 1901, Jackson[8] reviewed the European literature, and the earliest case reported was that by Durand-Fardel in 1837. Jackson found 13 cases in all, including one of his own. In 1911, Citelli[9] reported on 10 cases. In the same year, 1911, Trotter[10] described his classical triad for suggesting cancer of the nasopharynx: it consisted of impaired hearing, neuralgia of the second branch of the Vth nerve, and asymmetry of the soft palate. In 1913, Oppikofer[11] reviewed 250 cases with a detailed description of the signs and symptoms of nasopharyngeal cancer. In 1921, Regaud and Reverchon[12] reported on the pathologic features of lymphoepithelioma of the nasopharynx. In the same year, Schminke[13] reported a similar pathologic picture describing this same tumor.

New,[14,15,16,4,17] in 1921, 1922, 1925, 1931 and 1943, has successively reported on the greatest number of cases in the literature to date, and has contributed as much as any individual to the understanding and proper management of the disease.

In 1923, Crowe and Baylor[18] reported 52 cases. A few years later, in 1927, Quick and Cutler[19] first described the transitional cell cancer of the nasopharynx as a histopathologic entity. In 1928, Hansel[20] reported on 12 cases and reviewed the literature as a candidate's thesis for membership in the American Laryngological, Rhinological and Otological Society, Inc., just as the present author is doing 22 years later.

In 1929, Ewing[21] made the major contribution of a report on 110 cases with excellent histopathological differentiation.

In the 1930's, Dunlap,[22] C. L. Martin,[23] Furstenberg,[24] Kleinfeld,[5] Kasabach,[25] Hauser and Brownell,[1] Needles,[6] Digby,[26,27] and numerous others[28-36] began to fill the literature with various reports of cases.

In the 1940's, the outstanding papers among many were those of C. L. Martin,[37] H. E. Martin and Blady,[2] New and

Stevenson,[17] and Simmons and Ariel,[3] the latter being the most recent comprehensive report of 150 cases.

The author's present series includes patients treated at the Massachusetts General Hospital, Massachusetts Eye and Ear Infirmary, and Pondville State Hospital from 1930 through 1947; also patients treated at the Collis P. Huntington Memorial Hospital from 1930 until the hospital closed on Jan. 1, 1942. This series includes 102 cases.

ANATOMY INCLUDING LYMPHATIC DRAINAGE.

In order to appreciate fully the significance of numerous signs and symptoms that may suggest nasopharyngeal cancer, an anatomical perspective must be achieved.

Briefly, the nasopharynx may be described as a six-walled compartment. Its "anterior wall" is composed of the posterior portion of the nose, including the septum and the choanae through which the posterior tips of the middle and inferior turbinates project. One might also consider that it includes a portion of the soft palate along its inferior margin. The "floor" is the opening into the oropharynx, but on swallowing, the soft palate sweeps back to the posterior pharyngeal wall to form such a true floor. On the "posterior wall' the mucosa covers the fascial attachment of the superior constrictor muscle as it lies over the anterior portion of the basilar process of the occipital bone where it joins the sphenoid bone. On the "roof" of the nasopharyngeal cavity, the mucosal wall covers the main body of the sphenoid bone. The "lateral walls" are penetrated by the Eustachian tubes; surrounding the tubal orifice in a posterosuperior plane is the rounded eminence called the torus tubarius. The fossa formed by the torus and the actual lateral wall is the well known fossa of Rosenmüller, a very common site for the origin of nasopharyngeal cancer. The adenoid usually begins centrally on the posterior wall and extends up to and includes a portion of the superior wall, or the roof. An important adjacent structure is the foramen lacerum through which the internal carotid artery passes into the skull. The foramen lacerum is located about 1 cm. anterior to the fossa of Rosenmüller and

has been repeatedly shown to be a direct pathway for the local extension of tumor into the intracranial cavity; also, about 1 cm. lateral to the foramen lacerum are the foramen ovale and the foramen spinosum where still further direct intracranial extension can occur.

The mucosa covering the nasopharynx is composed of pseudostratified ciliated columnar epithelium which changes to stratified squamous epithelium as it approaches the oropharynx. There are occasional collections of mixed serous and mucoid secreting gland cells from which the occasional adenocarcinoma or even mixed tumor may arise. Beneath the epithelial layer in the corium are numerous lymphoid follicles from which small efferent ducts evolve.

Since in so great a number of cases cervical metastasis is the presenting symptom, the knowledge of lymphatic drainage of the nasopharynx is of importance. The lymphatic ducts begin in the adenoid and lymphoid tissue of the nasopharynx. Some of these join and descend inferiorly to the lateral and medial retropharyngeal nodes. Others pass laterally directly into the neck and to the upper deep cervical glands which are located just inferior to the superior attachment of the sternomastoid muscle. Some of the efferents from the retropharyngeal nodes also often extend to the deep cervicals. Various combinations may, of course, occur. Any of these efferents may then descend along the internal jugular vein where they form the jugular chain of nodes, and these in turn may connect with the supraclavicular region where is found the supraclavicular group of nodes. Efferents from the nodes of the upper deep cervical glands run along the anterior border of the trapezius muscle, following the course of the spinal accessory nerve, enter and then leave this so-called spinal accessory group of nodes to join with the supraclavicular nodes.

Thus the important lymph node groups are: *1.* the retropharyngeal nodes, medial and lateral; *2.* the upper deep cervicals; *3.* the internal jugular chain; *4.* the spinal accessory group; and *5.* the supraclavicular group.

HISTOPATHOLOGY.

Nasopharyngeal tumors as a group are just as varied as tumors in other parts of the body, but specific types of neoplasm are more prone to develop in the nasopharynx. As a general rule it might be said that most tumors of the nasopharynx are malignant. Stout[38] found 34 different tumor types in a review of the literature, including those in his own series. He reported from the records of the Presbyterian Hospital, of New York City, on 82 cases of nasopharyngeal tumors, of which 23 were benign and 59, or 72 per cent, malignant.

Among the primary malignant tumors of the nasopharynx in order of frequency Stout[38] found various grades of differentiation of epithelioma the most common, then lymphoepithelioma, lymphosarcoma, occasional plasmocytoma, fibrosarcoma and malignant teratoma. Among the secondary malignant tumors he mentions chordoma, craniopharyngeal duct tumors, leucemia, Hodgkin's disease and Boeck's sarcoid.

There has been considerable confusion throughout the literature regarding the nomenclature of the nasopharyngeal neoplasms which persists today in the various medical centers. As tumors of varying terminology with similar descriptions are placed before the otolaryngologist he must accept them as such for the present. Because of this problem, Salinger and Pearlman,[39] in 1939, in a report of 24 cases stressed the variation in the histopathology. They had three pathologists review all of their cases. They found the so-called transitional cell carcinoma as described by Quick and Cutler[19] as predominant in 75 per cent of their series; lymphoepithelioma was diagnosed in only six of their cases; however, pathologists still change the terminology and 15 years after their report confusion still abounds.

F. W. Stewart has been quoted by H. E. Martin and Blady[2] as employing the following basic differentiation of the epithelial tumors of the nasopharynx: the term squamous carcinoma includes those tumors with definite keratinizing tendencies; the transitional cell carcinoma included the type in

which the picture suggests duplication of the intermediate layer of the stratified epithelium. The term lymphoepithelioma is more difficult to differentiate because many of these tumors are really lymphosarcomas and are usually those in which the epithelial elements and the lymphoid elements have both undergone anaplastic change.

In our series the following histopathologic diagnoses were reported by the pathologists in the institutions from which our cases are recorded:

DIAGNOSIS BREAKDOWN.

	Total	Five-Year Survivals
Epidermoid carcinoma	49	1
Lymphoepithelioma	8	1
Undifferentiated carcinoma	4	1
Lymphoma	5	1
Plasmocytoma	4	—
Reticulum cell sarcoma	2	1
Adenocarcinoma	1	—
Lymphosarcoma	1	1
Transitional cell carcinoma	17	3
Embryonal carcinoma	1	—
Carcinoma (unclassified)	2	—
Carcinoma (clinical diagnosis)	8	2
	—	—
	102	11

The above chart demonstrates the great variety of histopathologic reports and why C. L. Martin,[23] in 1939, stated that "fortunately all the malignant neoplasms in the pharynx are very radiosensitive and their histopathology is not of any great clinical importance." In 1943, he qualified this[37] and stated that "85 per cent of the nasopharyngeal malignancies fall into the radiosensitive group including undifferentiated epithelioma, lymphoepithelioma, lymphosarcoma and plasmocytoma. Adenocarcinoma and well-differentiated epithelioma,

spindle cell cancer and the more differentiated chordoma, respond poorly to the doses of radiation usually effective in the former group."

Two tumor types found only occasionally in the nasopharynx deserve a more detailed description of their histology and life history. One, the chordoma, has already been well described by Furstenberg.[24] This is a tumor which has its origin in the remaining notochordal cell. It is most common at either extremity of the spinal column and arises in the vertebrae. Furstenberg reviewed the literature and found 52 cases reported, of which only 20 were located in the region of the nasopharynx. These tumors are of low malignancy but because of their location can rarely be removed surgically due to obvious technical difficulty.

The other tumor, the so-called nasopharyngeal fibroma, has also been well described in the past. It is an "adolescent" tumor occurring only between the ages of five and 25, after which it tends to disappear spontaneously; however, by its location it can act as a malignant lesion because of the pressure caused by its expansion. It is a smooth, solid tumor which is composed of fibroblasts, reticulum fibres which give it a firmer consistency than the true choanal polyp, and it contains a great many capillary blood vessels. It is highly vascular and even a biopsy may result in serious hemorrhage. The present treatment consisting of electrosurgical removal followed by radon seed implantation has been found extremely effective. The author has noted 11 cases in a 17-year review; all of these patients are alive and well and free of the tumor. These cases ranged from 11 to 21 years of age, there being 10 males and one female.

Plasmocytoma is considered of doubtful malignancy by Stout.[38] It tends to recur locally and often cannot be distinguished from the multiple myeloma of the plasma cell type. Malignant teratoma and dermoid cysts appear at birth or shortly thereafter — a good diagnostic point — and are very rarely found in the nasopharynx.

INCIDENCE: AGE, SEX AND RACE.

Nasopharyngeal cancer is not very common, but it occurs often enough, and its early diagnosis is made infrequently enough to warrant its emphasis. Nasopharyngeal cancer has been reported by Hertzfeld, according to Lack,[40] in 1906, as one case in 28,000 cases of cancer. He also stated that Finder calculated five cases in 40,000 and Gurlt, 18 cases in 10,000.

H. E. Martin and Blady[2] reported 2 per cent of all the malignant cases in the head and neck clinic at the Memorial Hospital in New York City to be nasopharyngeal cancers. At Pondville State Hospital, from 1930 to 1947, among a total of 13,578 malignancies, 27 were nasopharyngeal cancers, or an incidence of 0.002 per cent.

In the author's series the average as well as the median age is 49. The youngest case was age five, the eldest, 86. There are 20 patients under 40 years of age, 53 patients between 40 and 59 years, and 27 patients 60 years or over. In two cases the age is unknown or unstated.

The majority of nasopharyngeal tumors in our series as well as in others was found in males, the ratio being 76 males to 26 females.

In the past two decades mention has been made of the frequency of nasopharyngeal cancer in the Chinese. Digby, Thomas and Tse,[26] in 1930, in an outstanding article with detailed anatomic and histopathologic sketches, reported on 103 cases. They stated that in their 42-bed service in a Hong-Kong hospital it was not unusual to have four or five cases of cancer of the nasopharynx at one time. Dunlap,[22] in 1938, reported having treated 16 patients in six years of private practice, all of them either Chinese or other Orientals. He made mention of the fact that 40 per cent of his private patients were Chinese. He reported that Dr. Lois Green "writes from Changsha that one of her students found that the incidence of nasopharyngeal tumors there is 16 out of 336

cases." In our series it is of interest to note that there are four Chinese patients, or 3.9 per cent of those reported.

PATHOGENESIS AND SYMPTOMATOLOGY.

In view of the varied clinical findings and actual structural involvement, the resulting symptoms of the disease must be considered in some detail. It is in this particular area of the problem of cancer of the nasopharynx that the clinician must be well grounded, whether he be the internist, general practitioner, ophthalmologist, neurologist or otolaryngologist.

The diagnosis may be difficult to establish on first examination for many reasons. It may be nonapparent grossly on close, careful scrutiny; it may be represented as a slight fissure in the fossa of Rosenmüller; the first awareness of its presence may be the clinical finding of upper deep cervical lymphadenopathy. The latter is usually investigated by biopsy excision and may be found to contain transitional cell cancer or lymphoepithelioma, typical of a primary lesion in the nasopharynx; even then, repeated study of the nasopharyngeal vault might show no gross pathology. Often, following the initiation of radiation therapy to the glands, including the nasopharynx in the field, there will occur a breakdown in the superficial mucosa of the nasopharynx and the submucosal tumor then may be seen for the first time.

As the primary lesion enlarges in the nasopharynx it may encroach upon the Eustachian tubal orifice and produce unilateral blocked feeling in the ear, tinnitus, sensation of fluid in the ear, decreased hearing, and occasionally slight dizziness. At this time the drum is usually retracted. Purple to clear yellow serum may be noted through the drum in the middle ear, and the hearing loss is of a conduction type. Persistent repeated secretory otitis media may suggest the presence of a nasopharyngeal cancer involving the Eustachian tube.

The tumor as it enlarges may encroach upon the soft palate and produce a "nasal" voice. Increased postnasal discharge, occasionally bloody, soon ensues. After the tumor extends to

the choanae, unilateral nasal obstruction and increased nasal discharge, occasionally bloody, takes place. Repeated epistaxis may occur as a single symptom. If secondary infection occurs at this time a unilateral pansinusitis with its associated symptoms may develop.

The tumor may extend inferiorly and involve the lateral and medial retropharyngeal nodes, producing a lumpy feeling in the upper portion of the throat, associated with some soreness and even pain behind the palate, with referred pain to the ear on that side.

The tumor may then extend anterolaterally and involve the foramen lacerum and then continue on intracranially, pushing the dura ahead of it, actually (though rarely) penetrating the dura only when infection or trauma are superimposed. Marked headache may now become a symptom. The VIth nerve passes directly over the foramen lacerum and is frequently the first cranial motor nerve involved. The IIIrd, IVth and Vth cranial nerves lie just above the VIth nerve and as a result are the next to be involved. This situation would produce diplopia, due to internal strabismus, and parasthesias and pain along the branches of the Vth nerve, such as in the teeth, cheek and nose. As the sympathetic nerve chain about the internal carotid artery in the foramen lacerum is involved, Horner's syndrome with its miosis and enophthalmos occurs. Involvement of the superior cervical ganglion in the neck by direct extension from the internal jugular chain of involved cervical nodes can produce this same syndrome. The tumor may then extend from the foramen lacerum to the carotid groove and then to the supraorbital fissure causing exophthalmos, more headache, possible vomiting, choked discs, possible optic nerve atrophy, and finally complete ophthalmoplegia may occur.

Schlivek,[41] in 1937, stressed the ophthalmic symptoms he noted in a series of 38 cases. He emphasized that "one can evolve an ocular syndrome of nasopharyngeal tumor: involvement of the Vth, sympathetic, and VIth nerves, either indi-

vidually or together, with symptoms referable to the ear, are strongly suggestive of a nasopharyngeal tumor." Of his 38 cases, 16 had symptoms referable to the eyes. It can be said that once ocular signs occur, the tumor has already extended intracranially except in the case of Horner's syndrome, which is produced by involvement in the neck.

The tumor may extend laterally and anteriorly from the nasopharynx, entering the Eustachian tube to extend to the middle ear. Mastoiditis has occurred from superimposed infection and operations have been done on the mastoid on numerous occasions without the operator even being aware of the presence of a primary tumor in the nasopharynx. Deafness, middle ear discharge, often bloody, otalgia, and a pulsating tinnitus may develop from this extension of the tumor.

If the tumor should infiltrate along the pterygoid process and involve the internal pterygoid muscle, severe trismus occurs. In such a case described by Moyle[42] this was the presenting complaint of a 21-year-old man.

When the tumor extends anterolaterally and directly involves the levator palatini muscle an asymmetry of the soft palate is produced. Difficulty in swallowing and nasal voice may develop. A bulging of the palate should certainly strongly suggest this lesion.

Occasional extension through the bone of the base of the skull does develop but is less usual than extension via the natural foramina. Bach *et al*[43] reported a case with true extension through the sphenoid bone that continued superiorly and posteriorly to the petrous tip and eventually laterally to involve the internal carotid artery with perforation and fatal nasopharyngeal hemorrhage. True extension can result from stripping of the nutrient periosteum by the tumor, followed by bone infiltration *per se* by the tumor. Simmons and Ariel[3] observed this in six postmortem cases.

In 1942, Lenz[44] reported on a series of bone involvements demonstrated by X-ray. He found the tumor had involved the

body of the sphenoid in 10 instances, the greater wing of the sphenoid in 12, the petrous tip in 10, the basiocciput in eight, the foramen ovale and spinosum in five, the posterior margin of the vomer in three, and the sella turcica in three.

At one time or another all of the cranial nerves have been reported to be involved by this disease, but the VIIIth nerve and 1st nerve are by far the rarest. Martin and Blady[2] report the most common order of nerve involvement as the VIth, IIIrd, IVth, Vth, VIIth and IInd. The IXth, Xth, XIth and XIIth cranial nerves may become involved, mainly by extension from the metastatic glands in the neck. If the upper deep cervical nodes enlarge and become confluent with the upper group of the internal jugular chain, extension may take place up to the jugular bulb and then involve the cranial nerves that come through the jugular and hypoglossal foramina, namely, the IXth, Xth, XIth and XIIth. The jugular bulb syndrome of Jackson develops, producing hoarseness due to vocal cord paralysis, palatal paralysis and nasal regurgitation of food, a unilateral paresis and deviation of the tongue, and even some shoulder drop.

Ballenger[45] reported a case which produced VIIIth nerve paralysis by true extension to the petrous with encroachment by the tumor on the internal auditory meatus and involvement and destruction of the VIIth and VIIIth cranial nerves.

The VIIth nerve is more often involved peripherally by the same process that extends from the metastatic glands to the jugular bulb, destroying the nerve near its exit from the stylomastoid foramen.

One can readily see the importance to the neurologist of the nasopharyngeal cancer syndrome in relation to cranial nerve involvement. In 1941, Brock[46] stated that "from the neurological viewpoint a clinical picture referable to unilateral implication of the cranial nerves issuing from the middle or posterior fossa, should make one search carefully for enlarged retropharyngeal or cervical glands, followed by repeat exami-

nation of the nasopharynx." The importance of such follow-up examination cannot be overemphasized.

DIAGNOSIS.

The difficulties encountered in examining various orifices and cavities of the body hinder the frequency and care with which these orifices and cavities are studied. The nasopharynx by location is accessible only if the examiner has been trained and continues to train himself in the use of the instruments available. The best office procedure is the indirect method using the nasopharyngeal mirror, and supplemented by the use of the nasopharyngoscope; however, any patient who consults an otolaryngologist, no matter what the complaint, deserves a nasopharyngeal examination with one or both of these methods. The use of the Haslinger soft palate retractor followed by mirror examination is helpful in many cases. Under ether examination the Yankauer nasopharyngeal speculum is of considerable aid in the visualization of the postnasal space. Under general anesthesia the Lothrop soft palate retractor has proven a most satisfactory instrument for exposure of the nasopharynx. The use of the finger for palpation, which is distinctly distasteful to the patient, should be used only as a supplementary procedure under general anesthesia.

The author's office experience has been entirely satisfactory with use of the mirror and nasopharyngoscope. It is a rare nasopharyngeal lesion that will escape detection when these two methods are employed.

Not infrequently experienced otolaryngologists find it difficult to determine on careful gross examinations, the significance of adenoid-like tissue in the adult nasopharynx. The adenoid tends to disappear from adolescence onward, but in more and more adults persisting adenoid tissue has been noted. This is especially observed with the greater use of the nasopharyngoscope. Repeat observation frequently is urged and if doubt still persists, the tissue should be removed for biopsy.

Occasionally examination of the membrana tympani can give a clue to the presence of serious trouble in the nasopharynx. No Eustachian tube catheterization or even Politzerization should be attempted without prior visualization of the nasopharynx. Such visualization of the nasopharynx may lead to more frequent early diagnoses.

Recently Morrison[47] *et al.* have undertaken serious and fruitful cytological studies of the nasopharynx by smear technique. Their results have been most promising and are offered by them as an adjuvant means of arriving at a diagnosis. Although their results have been excellent, they admit that well trained technicians are necessary in order to make the diagnosis and screen the unquestionably negative slides.

Finally, X-ray studies of the nasopharynx must be fully utilized, particularly in all doubtful cases. The Roentgenologist well trained in the anatomy of the nasopharynx and its relation to the bones of the skull can be invaluable to us in helping to establish a diagnosis. Slight submucosal swellings in the vault may not be apparent to the examiner, despite use of the above methods of approach, but an accurate description of a small abnormal soft tissue swelling by the Roentgenologist may force the examiner into further action sooner. A bony defect may be of help seen by X-ray but usually indicates that the prognosis is very poor.

With use of the above techniques many more cases of cancer of the nasopharynx can be uncovered in the early stages of the disease.

INITIAL COMPLAINT.

As a rule what brings the patient with nasopharyngeal cancer to the physician? The chief complaint no doubt varies in proportion to the individual's response to the extensive cancer education program. This program has increased in intensity in recent years, and although the patients are appearing earlier with some signs and symptoms, there has been an apparent lag in the physician's appreciation of these symptoms early in the disease process.

The chief or initial complaints of the patients in our series in the order of significance to the patient may be listed as follows:

CHIEF COMPLAINT (Signs or symptoms which brought patient to hospital)		OTHER SYMPTOMS (Accompanying symptoms of less concern to patient)	
Symptoms	No. of patients	Symptoms	No. of patients
Nasal obstruction	28	Nasal obstruction	15
Cervical adenopathy	23	Weight loss	14
Deafness	22	Nasal bleeding	12
Headache	17	Deafness	11
Nasal bleeding	14	Postnasal drip	11
Postnasal drip	12	Headache	10
Sore throat	12	Tinnitus	7
Facial pain	8	Dyspnea	7
Dyspnea	6	Sore throat	6
Neurological symptoms	6	Cervical adenopathy	6
Ear pain	4	Ear pain	5
Tinnitus	4	Malaise	3
Oral bleeding	3	Neurological symptoms	3
Diplopia	2	Hemoptysis	3
Weight loss	2	Facial pain	3
Local numbness, Esotropia, Ocular protrusion, Hoarseness, Cough	1	Diplopia	2
		Esotropia, Ocular protrusion, Blindness	1

In this specific disease, unfortunately, the sign or symptom first noted by patient or physician may not appear until the disease has reached a practically incurable stage, with present methods of therapy.

The delay in diagnosis must be overcome. This must be stressed again and again or the five-year cure rate will never be increased beyond its present low level.

The literature is filled with discouraging reports of delayed diagnoses. In 1922, Woltman[48] observed that only one out of 25 cases had been correctly diagnosed before admission to the Mayo Clinic. New,[15] in 1922, reported 21 out of 79 with symptoms referable to the eye, 29 with symptoms referable to the ear, 38 with symptoms referable to the nose or nasopharynx, and 51 with cervical adenopathy. In reporting his

194 cases in 1931, New[4] reported that 42 per cent of these patients had cervical lymphadenopathy on admission.

In 1936, Salinger and Pearlman[39] reported that about 80 per cent of their patients showed metastases on admission. In 1938, Furstenberg[24] reported that 60 per cent of his series of 40 cases had cervical metastases on admission. This contributed a great deal no doubt to his pessimistic attitude regarding the disease which led to his much quoted statement at that time, "My meager experience with 40 cases leads me to conclude that treatment is exceedingly futile and the prognosis hopelessly discouraging." Only one of his 40 patients was alive after two years. Most of his patients came to him 15 months after the initial sign or symptom was observed.

In 1941, Kasabach[25] reported on 57 patients from the Presbyterian Hospital, of New York City, and stated that 40 cases, or 77 per cent, showed cervical adenopathy on admission, and 30 per cent revealed X-ray evidence of invasion of the basilar bones and adjacent bony structures.

As recently as 1949, Simmons and Ariel[3] demonstrated statistically the worst aspect of present day delay and inaccuracy in the diagnosis of cancer of the nasopharynx in patients arriving at their hospital. In their 150 cases, 44 patients, or 29.3 per cent, had involvement of one or more cranial nerves on admission; 130 cases, or 86.6 per cent, had cervical metastases and of these 67, or 44.7 per cent, had bilateral cervical metastases. Of the 150 cases the correct diagnosis was not suspected by the referring physician in 92 cases, or 61.3 per cent. What is most revealing in their stimulating report is that the interval between the onset of symptoms and seeking of medical advice by the patient averaged 3.2 months, whereas there was an average lapse of 6.9 months between the first visit to the physician and the time the correct diagnosis was established. The element of time lag was present to a lesser degree in our reported series of 102 cases.

In further evaluating our series the following accepted stage classification was used:

TOTAL NUMBER OF PATIENTS, 102.

	No. of patients
Stage 1A (Primary case, without metastases when first seen here)	40
Stage 1B (Primary case, with metastases when first seen here)	44
Stage 2A (Secondary case, without metastases when first seen here)	1
Stage 2B (Secondary case, with metastases when first seen here)	17

Of this group of cases, 61 out of 102, or 59.8 per cent, had metastases when they reported for treatment.

The average duration of symptoms before the first examination was as follows:

AVERAGE DURATION OF SYMPTOMS BEFORE FIRST EXAMINATION.

STAGE 1A (2 unknown)	STAGE 1B (5 unknown)
5½ months	8 months

ALL PATIENTS IN ALL GROUPS

Average duration	9 months (8 unknown)
Shortest duration	1 month
Longest duration	4 years, 5 months

The *delay in treatment* was considered the lapse of time between the date of the first examination and the date on which an accurate diagnosis was made and the treatment inaugurated. A lapse of two months or less was not considered a delay.

In 14 of the 102 patients, treatment for cancer was not begun within two months of the first visit because accurate diagnoses had not yet been made. The average "delay" of these 14 patients was five and one-half months; the longest "delay" was one year and two months; the shortest "delay" was two months and one week.

Early diagnosis is of major importance in preventing further delay in treatment.

TREATMENT.

The treatment of neoplasms of the nasopharynx seems to be less controversial than that in many other forms of cancer.

Since most of the malignancies of the nasopharynx are radiosensitive, radiation has proved to be the treatment of choice.

The results of such treatment are directly related to the time-lag or "delay" in arriving at a correct diagnosis. Since the inception of its use in nasopharyngeal cancer the results of radiation therapy have varied. It is not the purpose of this reviewer to delve into a field that belongs to the much more capable and experienced radiation therapists; however, some discussion is indicated in order to interpret and evaluate the progress made in the treatment of the disease in the past two decades.

Originally attempts were made to eradicate the disease by electrosurgery. For example, in 1930, Digby, Thomas and Tse,[26] reporting on their therapy in the surgical unit of the Hong Kong Hospital, stated that all cases perished despite local "diathermosurgery." No deep X-ray was available to them at the time of that report. In a later article the same year, Digby[27] states "that after ligature of the left common and external carotid arteries, and excision of the left superior maxilla, the growth in the nasopharynx was destroyed by diathermy; and one week later 2,706 mg. hours of radium were applied. Two weeks later 3,102 mg. hours of radium were again administered." All radium was given in 6 mg. hour tubes. He foresaw some hope for the future with the use of radium. In his prophecy he was correct, for not long after occasional reports of five-year cures by men in various parts of the world began to trickle into the literature. As the radiation technique, mainly by external maneuvers, improved, a greater, but not remarkable, number of cures were reported.

The author's series of 102 patients were treated as a general rule in the following manner: For the most part the lesions were treated through lateral pharyngeal portals, cheek portals and occasional intraoral portals. M. Schulz[49] recommends a tumor dose calculated to a minimum of 5,000 r. as sufficient for the usually radiosensitive nasopharyngeal cancers. The patients in this series included many treated in the early 1930's, at which time as little as 3,000 r. tumor dose was the

prevalent treatment. The more recently treated patients received a primary course of at least 5,000 r. calculated tumor dose.

In 1938, Blady[50] described a special instrument for irradiation of neoplasms of the nasopharynx with radium directly applied to the tumor, which has been used in several medical centers since then, but not in our particular series. He has been of the opinion, as have several other radiation therapists, that "by a combination of external and intracavitary irradiation, which is less than the maximum doses of either, the desired degree of radiation effect may be produced in the nasopharynx."

Cutler and Buschke,[51] in 1939, advised doses of 7,000 to 8,000 r. (measured on the skin) for carcinoma or lymphoepithelioma of the nasopharynx, while recommending a total dose of about 6,000 r. for lymphosarcoma of the nasopharynx.

As has been stated before, details relating to portals, such as the intraoral approach, or lateral, anterior or posterior portals, as indicated, should be left in the hands of the radiologist who should work in conjunction with the otolaryngologist in order that the maximum effective tumor dose be directed to that area which contains the tumor itself.

Protection of the eyes during this therapy is an important factor and is provided for by the radiation therapist. Of great importance in our clinics has been the increasing attention directed to the teeth, whether they be in excellent or poor condition. It is our policy at this time to recommend the removal of all teeth in the proposed field of radiation regardless of their condition or the age of the patient, at least one week prior to the inauguration of radiation. The incidence of radiation osseonecrosis has been greatly reduced in our experience because of this precaution. This is especially valuable in the cases in which the intraoral portal is to be utilized.

Up to the 1920's practically all patients with nasopharyngeal cancer died. Coutard[52] did a considerable amount of pioneer work in improving the techniques of radiation ther-

apy. His results were compiled by DelRegato from L'Institute du Radium (1919-1931). Nine cases of carcinoma of the nasopharynx were treated and one case was well, after five years. In 1936, Kleinfeld[5] reported on 40 cases. All of these had died but two, who were but one year past treatment. The longest any of his cases lived was three years.

In 1936, Duhig,[31] of Australia, reported on the treatment of a 13-year-old girl with cervical metastases. He had achieved only palliative help with radium but was encouraged by its use.

In 1938, Furstenberg[24] reported that only one of his 42 cases was alive after two years. He was certainly discouraged at that time.

The same year, 1938, Hauser and Bramwell[1] reported only one out of their 50 cases was alive five years after treatment.

In 1940, H. E. Martin and Blady[2] showed a great change in the end-results. They reported the tremendous number of 20 five-year cures, or 25 per cent, in a series of 80 patients. The following year, in 1941, Figi[53] reported a 10-year cure in a child with highly malignant cancer of the nasopharynx with bilateral cervical metastases.

That same year, 1941, Kasabach[25] reported 14 cases (33 per cent) of 42 cases living three or more years, but only seven cases (16.6 per cent) of five-year cures.

In 1942, Lenz[44] reported a five-year cure in 27.6 per cent of a series of 44 cases. This report included all cases regardless of the stage of the disease.

New and Stevenson,[17] in 1943, reported on their extensive series of 271 cases, but these included cases treated when radiation therapy for these tumors was given in smaller doses than recommended today; in fact, all these cases were treated prior to 1937. They reported that out of 234 cases with positive biopsies, only 13 per cent were living after five years.

In 1949, Simmons and Ariel[3] reported the death of 71 out of 78 determinate patients, a net five-year survival rate of 9.8 per cent.

With today's radiation techniques, whether radium alone, external Roentgen rays alone, or a combination of both is used, it is difficult to prophesy that a certain percentage of five-year cures should be obtainable. At this time too many factors play a part to reduce the whole problem to one of percentage cure in relation to the number of cases; therefore, in reporting the end-results of the 102 cases in our series, it must be restated that these have been mainly collected from four major institutions. The type of patient admitted, the source of admission, and the examining techniques employed by the individual physicians directly connected with each case, produce as varied a result as do the reports of other clinics in the past.

FIVE-YEAR CURES

STAGE 1A

"Potential Survivors".....35
Five-Year Cures 10
28.6%

(Nineteen of the total have been treated in the past five years, therefore cannot be included in the Five-Year potential survivor group.)

THREE-YEAR CURES

STAGE 1A

"Potential Survivors" 37
Three-Year Cures 10
27%

(Nine of the total have been treated in the past three years, therefore cannot be included in the Three-Year potential survivor group.)

(N.B.—All patients in this stage who survived three years also survived five years. Two potential survivors have lived three years but not five years since treatment was begun.)

STAGE 1B

"Potential Survivors 32
Five-Year Cures 1
3.1%

STAGE 1B

"Potential Survivors" 40
Three-Year Cures 2
5%

(Eight potential survivors have lived three years but not five years since treatment was begun.)

Total Stage 1A and 1B.....16.4%

Total Stage 1A and 1B.....15.7%

ALL STAGES (1A, 1B, 2A, 2B)

"Potential Survivors" 83
Five-Year Cures 11
13.3%

ALL STAGES (1A, 1B, 2A, 2B)

"Potential Survivors" 93
Three-Year Cures 12
13%

(Seven of the total number of patients cannot be traced and so are considered dead of the disease.)

It is of striking importance prognostically from the results of our series, that if a patient with a nasopharyngeal cancer in Stage 1A, who has been "adequately" treated, should reach the three-year limit without recurrence, his chances for survival would then be excellent. The 10 three-year cures were also the 10 five-year cures. It is interesting to go back to a statement by Munro Black, quoted by Capps,[54] in 1939. Black,[55] in a Hunterian Lecture, stated that "if a good local result is obtained and the patient passes the three-year limit without distant metastasis, the outlook would appear good. If treated, or only treated late in the disease, most seem to run a fatal course in under two years." The author's statistical survey appears to bear this out.

LOCAL RECURRENCE AND METASTASES.

Cases which recur after primary treatment must still be managed by the physician. The question of more palliative radiation or surgical intervention must be considered from time to time. Neurosurgical procedures for relief of pain have been effective in many cases.

In this series the following time intervals are of interest in relation to recurrence in the various stages:

LOCAL RECURRENCE (Average time lapse after initial treatment)

Stage 1A	1 year, 5 months
Stage 1B	8 months
Stage 2A and 2B	1 year, 6 months

LOCAL EXTENSION (Average time lapse after onset of symptoms)

Stage 1A	5½ months
Stage 1B	1¼ months

REGIONAL GLANDS (Average time lapse after onset of symptoms)

Stage 1A	2 years, 1 month
Stage 1B	3 years, 6 months

Distant metastases seem more common in nasopharyngeal cancer than in other respiratory tract cancer.

DISTANT METASTASES (Average time lapse after onset of symptoms) (Lungs, axilla, liver, spleen, cecum and bones)

Stage 1A	2 years, 3 months
Stage 1B	10 months
Stage 2A and 2B	2 years, 7 months

C. L. Martin[23] states that "generalized metastases appear in the chest, bones, liver, kidneys, spleen, and retroperitoneal nodes as in any type of disseminated malignant disease." H. E. Martin and Blady[2] reported dissemination of tumor below the clavicle in 34 per cent of their cases. In their series, in those cases with uncontrollable disease the average length of life was about 14 months.

In the 37 autopsies obtained from 128 deaths reported by Simmons and Ariel,[3] 20 patients, or 54 per cent, had generalized metastases.

The nasopharynx itself is rarely the site of distant metastasis. This occurred in a patient treated at the Massachusetts Eye and Ear Infirmary in whom metastasis was found in the nasopharynx from a primary neoplasm of the prostate.

SUMMARY.

A report of 102 cases of nasopharyngeal cancer has been given. These patients were seen and treated at the Massachusetts Eye and Ear Infirmary, Massachusetts General Hospital, Pondville State Hospital, and the C. P. Huntington Memorial Hospital before it was closed.

Of the 102 cases, there were 35 "potential survivors" — Stage 1A (primary case without metastasis when first seen) and 10 of these cases survived three years; and the same 10, or 28.6 per cent, survived five years without recurrence; therefore, all cases in this series that survived three years also survived five years.

In Stage 1B (primary case with metastasis when first seen here) there was but one five-year cure, or 3.1 per cent, of 32 cases.

All stages (1A, 1B, 2A, 2B) included 83 "potential survivors." (Nineteen of the total have been treated in the past three years and, therefore, cannot be included in the five-year "potential survivor" group.) Of these, only 11, or 13.3 per cent, of the entire series are five-year survivors.

The average duration of symptoms before the first examination in Stage 1A was five and one-half months, and in Stage 1B, eight months.

The five most common chief complaints of patients on admission were nasal obstruction, cervical adenopathy, deafness, headache and nasal bleeding.

Fourteen of these 102 patients were not treated for this disease within two months of the first visit because accurate diagnoses had not been made. (The lapse of time between the date when the patient was first examined and the date on which accurate diagnosis was made and treatment, therefore, inaugurated was considered as the "delay.") A lapse of two months was not considered "delay." The average "delay" in these 14 patients was five and one-half months.

CONCLUSIONS.

The results obtained in this series compare favorably with those of other reported series to date.

The problem of early diagnosis can be solved partially by more careful attention to the early signs and symptoms produced by the disease. The time-lag between onset of symptoms and accurate diagnosis must be shortened in order to give the patient a better chance for survival with the present radiation therapeutic techniques.

New's[15] statement of 1922 bears requoting 37 years later. "Malignant tumor of the nasopharynx is much more common than believed. The syndrome which it presents is not generally known. This accounts for the fact that many patients are treated medically and surgically without discovery of the tumor. There is a striking lack of nasal or nasopharyngeal symptoms in many of the cases."

The progress made during the last decade has proven that there is hope for combating this serious disease. Earlier diagnoses mean more survivors of nasopharyngeal cancer.

BIBLIOGRAPHY.

1. Hauser, I. J., and Brownell, D. H.: Malignant Neoplasms of the Nasopharynx. *Jour A. M. A.*, 111:2467-2473, Dec., 1936.

2. Martin, H. E., and Blady, J. V.: Cancer of the Nasopharynx. *Arch. Otolaryngol.*, 32:692-727, Oct., 1940.

3. Simmons, M. W., and Ariel, I. M.: Carcinoma of the Nasopharynx. *Surg., Gynec. and Obst.*, 88:763-775, June, 1949.

4. New, G. B.: Highly Malignant Tumors of the Nasopharynx and Pharynx. *Jour. Amer. Acad. Ophth. and Otolaryngol.*, 36:39, 1931.

5. Kleinfeld, L.: Malignancies of the Nasopharynx. The Laryngoscope, 46:415-418, June, 1936.

6. Needles, W.: Schmincke Tumor. *Jour. Mt. Sinai Hosp.*, 1:266-268, Mar.-Apr., 1935.

7. Smith, G. E., and Dawson, N. R.: Egyptian Mummies. London: George Allen and Unwin, Ltd., 1924.

8. Jackson, C.: Primary Carcinoma of the Nasopharynx: A Table of Cases. *Jour. A. M. A.*, 37:371, Aug. 10, 1901.

9. Citelli: Ueber 10 Fälle von Primären Malignen Tumoren des Nasenrachenraumes. *Ztschr. f. Laryngol., Rhinol. u. Otol.*, 4:331, 1911.

10. Trotter, W.: On Certain Clinically Obscure Malignant Tumors of the Nasopharyngeal Wall. *Brit. Med. Jour.*, 2:1057, 1911.

11. Oppikofer, E.: Ueber die Primären Malignen Geschwülste des Nasenrachenraumes. *Arch. f. Laryngol.-Rhinol.*, 27:256, 1913.

12. Regaud, C., and Reverchon, L.: *Rev. Laryngol. and Bord.*, 369-378, Regaud C. (quoted by Reverchon and Coutard): Lymphoepitheliome de l'Hypopharynx Traité Par la Roentgen Therapie. *Bull. et Mem. Soc. d'Otol., Rhinol. u. Laryngol. Congress*, March 9, 1921.

13. Schminke, A.: Ueber Lymphoepitheliale Geschwülste. *Beitr. z. Path. Anat.*, 58:161-170, 1921.

14. New, G. B.: The Relation of Nasopharyngeal Malignancy to Other Diagnoses. *Minn. Med.*, 4:419, 1921.

15. New, G. B.: Syndrome of Malignant Tumors of the Nasopharynx. *Jour. A. M. A.*, 79:10-13, 1922.

16. New, G. B.: Treatment of Malignant Tumors of the Pharynx and Nasopharynx. *Surg., Gynec. and Obst.*, 40:177, 1925.

17. New, G. B., and Stevenson, W.: End-Results of Treatment of Malignant Lesions of the Nasopharynx. *Arch. Otolaryngol.*, 38:205-209, Sept., 1943.

18. Crowe, S. J., and Baylor, J. N.: Benign and Malignant Growth of the Nasopharynx and Their Treatment with Radium. *Arch. Surg.*, 6:429, Mar., 1927.

19. Quick, D., and Cutler, M.: Transitional Cell Epidermoid Carcinoma. *Surg., Gynec. and Obst.*, 45:320, 1927.

20. Hansel, F. K.: Malignant Tumors of the Nasopharynx. *Arch. Otolaryngol.*, 9:12-22, Jan., 1929.

21. Ewing, J.: Radiosensitivity. *Radiol.*, 13:313, Oct., 1929.

22. Dunlap, A. M.: Malignancy of the Nasopharynx and Eustachian Tube. *Chinese Med. Jour.*, 53:68-71, Jan., 1938.

23. Martin, C. L.: Complications Produced by Malignant Tumors of the Nasopharynx. *Amer. Jour. Roentgenol.*, 41:377-390, Mar., 1939.

24. Furstenberg, A. C.: Malignant Neoplasms of the Nasopharynx. *Surg., Gynec. and Obst.*, 66:400-404, Feb., 1938.

25. Kasabach, H. H.: Symposium on Nasopharyngeal Tumors. The Laryngoscope, 31:459-463, May. 1941.

26. Digby, K. H.; Thomas, G. H., and Tse, H. S.: Notes on Carcinoma of the Nasopharynx. Caduceus (Univ. of Hong Kong). 1930, 9, May, No. 2.

27. Digby, K. H.: Nasopharyngeal Carcinoma Without Clinical Enlargement of Lymphatic Glands in the Neck. Caduceus, 1930, 9, Nov., No. 4.

28. Ball, C. E.: A Case of Carcinoma of the Nasopharynx. *Tex. State Jour. Med.*, Ft. Worth, 26:719-721, 1931.

29. Ch'eng, Y. L.: Lymphoepithelioma of the Nasopharynx with Involvement of the Nervous System. *Chinese Med. Jour.*, 49:1075-1091, Oct., 1935.

30. Duffy, J. J.: A Nasopharyngeal Tumor. *Jour. Med. Soc., N. J.*, 34: 605, Oct., 1937.

31. Duhig, J. V.: Lymphoepithelioma. *Med. Jour. Australia*, 2:81-82, July, 1936.

32. Guthrie, T., and Evans, W. H.: An Unusual Case of Endothelioma of the Nasopharynx. *Jour. Laryngol. and Otol.*, 47:406-409, June, 1932.

33. Heine, L. H.: Malignant Tumors of the Nasopharynx. *Arch. Otolaryngol.*, 22:51-61, July, 1935.

34. Hill, F. R.: Malignant Disease of the Sinuses and Nasopharynx in the Small Hospital. *Ann. Otol., Rhinol. and Laryngol.*, 46:158-168, Mar., 1937.

35. Tolan, F. F.: Malignant Neoplasm of the Nasopharynx. Va. Mason Hosp. Clin., Dec., 1939.

36. Weinberger, N. S.: Malignant Tumors of the Nasopharynx. *Ann. Otol., Rhinol. and Laryngol.*, 48:203-211, Mar., 1939.

37. Martin, C. L.: Cancer of the Nasopharynx. *Ann. Otol., Rhinol. and Laryngol.*, 52:146-160, Mar., 1943.

38. Stout, A. P.: Symposium on Nasopharyngeal Tumors. The Laryngoscope, 31:446-450, May, 1941.

39. Salinger, S., and Pearlman, S. J.: Malignant Tumors of the Epipharynx. *Arch. Otolaryngol.*, 23:149-172, Feb., 1936.

40. Lack, H. L.: The Diseases of the Nose and Throat and Its Accessory Sinuses. London: Longmans, Green and Co., 1906.

41. Schlivek, K.: Ocular Manifestations of Malignant Nasopharyngeal Tumors; Report of Cases. *Arch. Ophth.*, 17:1055-1072, June, 1937.

42. Moyle, E. H.: Nasopharyngeal Malignancy; Case Report. The Laryngoscope, 43:283-284, Apr., 1933.

43. Bach, A. S.; Lederer, F. L., and Palevsky, S. N.: Nasopharyngeal Carcinoma. *Arch. Otolaryngol.*, 31:529-539, Mar., 1940.

44. Lenz, M.: Roentgen Therapy of Primary Cancer of the Nasopharynx. *Amer. Jour. Roentgenol.*, 48:816, 1942.

45. Ballenger, H. C.: Carcinoma of the Nasopharynx with Destruction of the Cranial Nerves, Including the IInd and VIIIth Nerves. *Ann. Otol., Rhinol. and Laryngol.*, 43:899-902, 1933.

46. Brock, S.: Symposium on Nasopharyngeal Tumors. The Laryngoscope, 31:455-457, May, 1941.

47. Morrison, L. F.; Hopp, E. S., and Wu, R.: Diagnosis of Malignancy of the Nasopharynx. Cytological Studies by the Smear Technique. *Ann. Otol., Rhinol. and Laryngol.*, 58:18, Mar., 1949.

48. Woltman, H. W.: Malignant Tumors of the Nasopharynx with Involvement of the Nervous System. *Arch. Neurol. and Psych.*, 8:412, 1922.

49. Schulz, M.: Massachusetts General Hospital. Personal communication.

50. Blady, J. V.: A New Instrument for Irradiation of Tumors of the Nasopharynx with Radium or Radon. *Amer. Jour. Roentgenol.*, 40:723-729, Nov., 1938.

51. Cutler, M., and Buschke, F.: Cancer, Its Diagnosis and Treatment. Philadelphia, Pa.: W. B. Saunders Co., p. 144.

52. Coutard, H.: Roentgen Therapy of Epitheliomas of the Tonsillar Region, Hypopharynx and Larynx from 1920 to 1926. *Amer. Jour. Roentgenol. and Rad. Ther.*, 28:313, 1932.

53. Figi, F. A.: Highly Malignant Carcinoma of the Nasopharynx with Bilateral Cervical Metastasis in a Child—Well After 10 Years. *Ann. Otol., Rhinol. and Laryngol.*, 50:1218-1220, Dec., 1941.

54. Capps, F. C. W.: Lymphoepithelioma in the Nasopharynx. *Jour. Laryngol. and Otol.*, 54:133-140, Mar., 1939.

55. Black, Munro J. S.: The Lymphoepitheliomata. Hunterian Lecture. *Jour Laryngol. and Otol.*, Apr., 1938.

THE PERMANENTLY PATENT FENESTRA NOV-OVALIS.*

JULIUS LEMPERT, M.D.,
New York, N. Y.

Osteogenetic closure of the newly created fenestra following fenestration of the vestibular labyrinth for clinical otosclerosis has been the greatest deterrent to the permanent maintenance of the early hearing improvement obtained following this surgery. This has been recognized by otologists ever since this surgery has come into existence. This fact, though recognizable, was not surprising, since it was natural to expect nature to carry out its usual function of repair within the bony labyrinthine capsule just as it was expected to repair all the other surgically injured soft tissues.

We could hardly have expected nature to repair all injured tissue without repairing the newly created fenestra as well, since the identically existing constitutional biologic factors which govern repair following surgical injury in one type of tissue govern all types of injured tissue.

In recognition of this natural law of overall repair, in order to assure the permanent patency of the newly created fenestra, we were for the first time confronted with the problem of experimentally seeking means and ways of preventing nature from performing its naturally inherent duty of repair in the bony fenestra region without interfering with its mission of repairing the rest of the injured tissues.

With this mission in mind I decided first to observe and study how nature repairs the newly created fenestra, how it

*Read at the meeting of the Eastern Section, American Laryngological, Rhinological and Otological Society, Inc., Philadelphia, Pa., Jan. 12, 1951.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 18, 1951.

closes it by osteogenesis and where the osteogenesis starts and ends in a postfenestrated ear. I hoped thus, as a result of the knowledge gained from such a study, perhaps to be able to find ways and means of interfering with nature's mission of repair and preventing osteogenesis either from starting or at least from completing its mission if its start could not be prevented. In all surgery nature's law of repair is the greatest asset to the surgeon, but in fenestration surgery it is both the combined friend and enemy of the otologic surgeon. Here the surgeon expects the unnatural, he wants repair everywhere except within a bony area 2 mm. wide and 6 mm. long which is the fenestra gap created in the bony vestibular labyrinth. Isn't this asking too much of nature?

What have I learned from the above mentioned study and how was this knowledge applied in an attempt to try and serve our purpose of keeping osteogenesis from taking place? I have revised 517 osteogenetically closed postoperative labyrinthine fenestras and studied the observations made in these fenestras. I learned therefrom:

1. That bone-dust when left in the region of the fenestra stimulates and hastens its closure by osteogenesis. I, therefore, advocate that all bone-dust be meticulously removed.[1]

2. That the most meticulous removal of bone-dust does not prevent ultimate bony closure of the fenestra. Fenestras created without the burr and, therefore, without formation of bone-dust, closed as a result of osteogenesis as well.

3. That osteogenesis does not as a rule take place underneath the tympanomeatal flap on the outer surface of the bony labyrinthine capsule in the region surrounding the newly created labyrinthine fenestra.

4. That osteogenesis takes place underneath the tympanomeatal flap as a rule only intramarginally within the fenestra gap in the fenestra rim.

5. That osteogenesis of the fenestra rim as a rule begins within the innermost endosteal bony layer. I did not fail to realize, however, that as far as the end-result was concerned

it made no difference whether the periosteal layer when not removed also regenerated or the enchondral layer left intact did not regenerate; neither would it make any difference if both of these bony histologic layers were removed to prevent them from participating in the osteogenetic process, since osteogenesis of the endosteal layer alone within the fenestra rim is sufficient to result in fenestra closure.

In the hope of preventing osteogenesis within the endosteal layer of the fenestra rim I advocated burnishing the fenestra rim with a gold burnishing burr which was proven experimentally in the animal to delay osteogenesis but not to prevent it.[2] As a result of further experimentation with burnishing the fenestra rim with various metals, I found that burnishing the fenestra rim with lead in two Rhesus monkeys prevented osteogenesis[3] and used the lead-burnishing of the human fenestra rim since; however, despite the fact that the percentage of closures was reduced since its use, I never knew which fenestra would remain open and which one would not.

I was never able to prove to myself or anyone else that the fenestra which remained open did so because of having been burnished with lead. I was not unmindful of the fact that many fenestras which I did not burnish with lead remained open as well, nor did I overlook the fact that many otologists who were not using lead were also rewarded with a fair percentage of permanently open windows following this surgery; however, they also were unable to prove either to their own or anybody else's satisfaction why these fenestras remained patent while other fenestras which they made with same technique closed by osteogenesis.

I succeeded in preventing bone regeneration within the fenestra rim by physically impeding osteogenesis with metal obturators; however, I found it technically difficult to make a fenestra which could be snugly fitted with a prefabricated prosthesis.[4] To make a prosthesis to fit individually each newly created fenestra was found not to be practical. I found that I could improve hearing and prevent osteogenesis within the fenestra rim with a properly inserted mobile cartilage

stopple;[5] however, in a large percentage of cases the ultimate formation of fibrous tissue between the fenestra rim and the stopple eventually rendered the stopple immobilizable by air-borne sound, and I had to abandon the use of the cartilage stopple.

6. I have learned that osteogenesis took place less frequently in the region nearest the ampulla of the external semicircular canal. I thought this was so because the fenestra was as a rule wider in this region. As a result of this observation I developed and described, in 1941, the fenestra nov-ovalis technique,[4] whereby a much wider fenestra is created in the surgical dome of the vestibule. This new technique was universally adopted since. Otologists employing the fenestra nov-ovalis technique have since observed and reported a much larger percentage of permanently patent fenestras than following their use of my originally described one-stage technique whereby the fenestra was created in the narrow portion of the external semicircular canal posterior to the ampulla; however, this universal observation has, nevertheless, failed to explain why and how some fenestras made with my originally described one-stage technique[6] which could not be made as wide and were much narrower remained permanently patent. It also failed to explain why, despite the use of the fenestra nov-ovalis technique, about 30 per cent of the fenestras closed, nevertheless, as a result of osteogenesis.

7. I learned that osteogenesis sometimes takes place within the perilymph space, due to bone chips unavoidably lost within it, following the pulverization of the bony endosteal layer of the labyrinthine capsule. As a result of this knowledge gained I have developed and since employed the bone-dust-free cupola technique[7] for creating the fenestra. Bone-dust no longer being formed with this technique, the problem of its removal has been done away with, and perilymphatic osteogenesis can thus be avoided; however, this bone-dust-free technique was not expected to and does not prevent osteogenesis from taking place in the freshly injured fenestra rim margins.

In summarizing my above mentioned study of osteogenetic fenestra closure and the problem of its solution, it is not dif-

ficult to see that despite the fact that I have learned much about how a newly created window closes as a result of osteogenesis and succeeded in greatly reducing the percentage of closures by developing and employing the fenestra nov-ovalis technique, I, nevertheless, failed to learn therefrom:

1. Why osteogenesis was prevented from taking place in a much larger percentage of ears following the use of the fenestra nov-ovalis technique?

2. Why osteogenesis was not prevented from taking place in all the fenestras following the use of the fenestra nov-ovalis technique?

3. Whether the creation of a wider fenestra was the sole reason for the prevention of osteogenesis in these patients whose fenestras remained patent, or was it only a contributory factor to its prevention by some other still unknown main factor?

4. What was the main factor without the existence of which osteogenesis could not be and was not prevented within the fenestra rim, even in the presence of a wider fenestra?

5. When osteogenetic repair took place why did it do so only intramarginally within the fenestra gap in the fenestra rim and not extramarginal to the fenestra gap outside the fenestra rim, regardless of whether the fenestra was made wide as in the fenestra nov-ovalis technique or narrow as in the originally employed one-stage technique?

Since some windows closed and others stayed open with use of all the media, within my knowledge, employed for the prevention of osteogenesis, it occurred to me that perhaps none of these media are capable of preventing it, and that some other as yet nonidentified factor is at work when osteogenesis is prevented. Though osteogenesis was prevented from starting in a much greater percentage of fenestra rims following the use of the fenestra nov-ovalis technique, I was, nevertheless, not convinced that making the fenestra wider was the sole reason for preventing the start of osteogenesis within the freshly injured rim margins of the fenestra gap in a much

larger percentage of postoperative fenestras than heretofore obtainable in narrow fenestras. I could envisage that a wider fenestra may occasionally not complete its mission of fenestra closure after osteogenesis started but could not understand why the creation of a wider fenestra should prevent osteogenesis from starting in its freshly injured fenestra rim.

It, therefore, occurred to me that the same still nonidentified factor which prevented osteogenesis in some of the narrow fenestras was most likely also the one which prevented it in the wider fenestras. Perhaps the reason why more of the wider fenestras stayed open was because a wider fenestra favored this main factor more frequently. I still further reasoned that if there is such a factor, and if a wider fenestra favors the prevention of osteogenesis by this factor, then why did it not prevent it in every one of the fenestras created with the fenestra nov-ovalis technique? This suggested further that perhaps this factor unknown to me did not always exist and in its absence bone regeneration did take place.

To seek an answer to these questions, I had to resort to studying the permanently patent fenestra following the fenestra nov-ovalis technique and also following my first described original technique to see if the same factor prevails in both techniques. I thought that by studying the fenestra gap and rim of the permanently patent fenestra I might be able to recognize and observe some factor still unknown to me which might be held responsible for the prevention of osteogenesis in the fenestra rim. I also hoped that the findings in the fenestra gap of the patent fenestra may perhaps also lead me to the recognition of the factor responsible for the consistent prevention of osteogenesis outside the fistula.

I investigated the fenestra region in 100 ears which failed to show improved hearing following fenestration, in order to observe if possible how osteogenesis was prevented from taking place in some of the fenestra rim margins and why it was not prevented in the others. I wanted to see if there is any difference outside of observable osteogenesis between a closed and an open window.

After studying 100 postoperative fenestras of ears which failed to show improvement in hearing following the fenestra nov-ovalis operation, I found 28 of them closed by bone regeneration and 72 fenestras permanently patent because bone regeneration did not take place. In the 28 osteogenetically closed fenestras the findings were the same as in the 517 previously examined closed fenestras. The flap was readily lifted and separated from its attachment to the outer surface of the fenestrated region. No bone regeneration has taken place in this region. The fenestra gap was found to be obliterated by newly regenerated bone. The inner surface of the entire flap

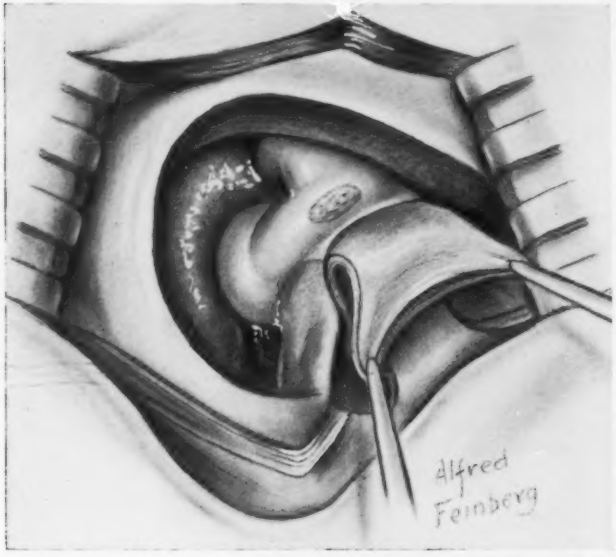


Fig. 1. Smooth inner surface of tympanomeatal flap as seen during revision of every osteogenetically closed fenestra.

was found to be evenly smooth as it was when originally placed to cover the fenestra region (see Fig. 1). This held true whether fenestra region was gold-burnished or lead-burnished or not burnished at all.

In every one of the 72 fenestras which remained patent because osteogenesis did not take place, either extramarginal to the fenestra rim or intramarginally in the rim of the fenestra gap, the following significant differentiating observations were made: The tympanomeatal flap was found to be not only adherent to the bony labyrinthine capsule extramarginal to the fenestra gap, but the inner surface of the portion of the flap facing the fenestra gap was found to be invaginated into the fenestra gap and adherent intramarginally to the bony fenestra rim. After slowly and gently separating the flap from its adhesion intramarginally to the bony

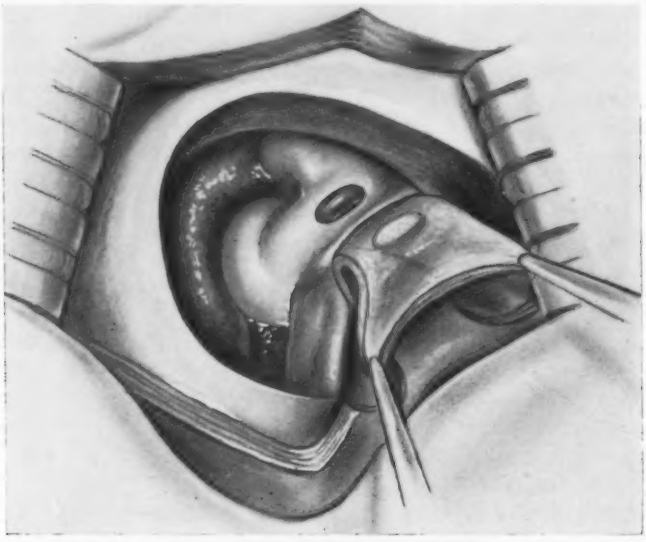


Fig. 2. Moulded impression of fenestra gap on inner surface of tympanomeatal flap as seen during revision of every permanently patent fenestra.

fenestra rim and lifting it from the entire outer surface of the bony capsule extramarginal to the fenestra gap, a moulded impression of the fenestra gap was observed to exist on the inner surface of the tympanomeatal flap in every one of the 72 patients in whom the fenestra remained patent (see Fig. 2). The membranous labyrinth which was apparently not

adherent to the flap remained undisturbed in its position within the perilymph space. In other words, whenever invagination of the flap into the fenestra gap and intramarginal adhesion of its inner surface to the bony fenestra rim was observed to have taken place, osteogenesis within the bony

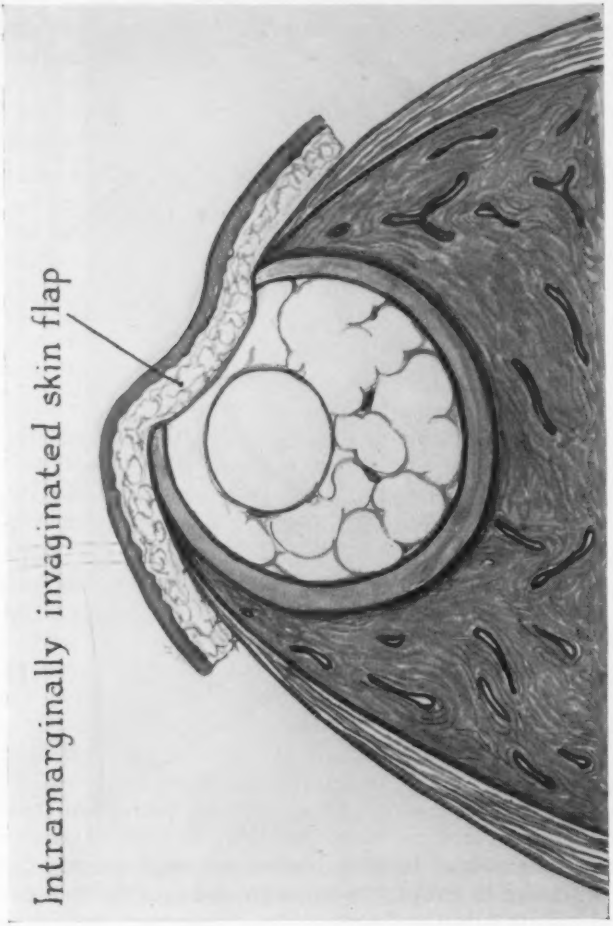


Fig. 3. Diagram Showing Cross-Section of Permanently Patent Fenestra. Whenever the inner surface of the tympanomeatal flap became invaginated into the fenestra gap and became adherent to the lateral walls of the fenestra rim osteogenesis was prevented intramarginally within the fenestra rim and fenestra remained permanently patent.

fenestra rim was prevented and permanent patency of the fenestra resulted (see Fig. 3). On the other hand, whenever

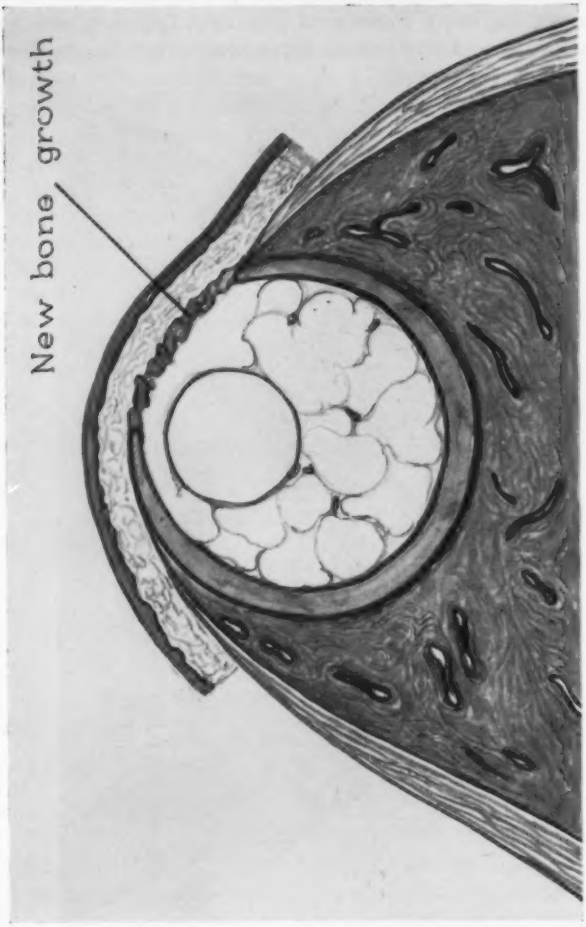


Fig. 4. Diagram Showing Cross-Section of Osteogenetically Closed Fenestra. Whenever the inner surface of the tympanomeatal flap did not become invaginated into the fenestra gap and it did not become adherent to the lateral walls of the fenestra rim, osteogenesis started intramarginally within the fenestra rim and eventually closed the fenestra gap.

the flap was observed to have become adherent outside the fistula but failed to have become invaginated into the fenestra gap and adherent to the fenestra rim, osteogenesis was pre-

vented only outside the fistula and not in the fenestra rim (see Fig. 4).

Since my May, 1950, report before the American Otological Society[8] I have revised five fenestras in patients who enjoyed practical hearing following fenestration for periods varying from three to seven years, but suddenly lost their hearing improvement and became worse without ever regaining their hearing. The fenestra was found to be completely patent and free from osteogenesis in all five cases. The observations made on the operating table were exactly the same as those made in the other 72 permanently patent fenestras.

The above mentioned observations led me to the conclusion that no matter what I, heretofore, did or did not do to prevent osteogenesis from starting in the fenestra rim and eventually closing the fenestra gap following the fenestration operation, unless invagination of the flap accidentally took place and its inner surface became adherent to the fenestra rim within the fenestra gap, osteogenesis took place and closed the fenestra gap.

The ultimate fate of the newly created fenestra was nonpredictable because osteogenesis evidently was prevented only when the pressure exerted by the mastoid wound packing accidentally happened to be sufficient to result in invagination and adhesion of the flap to the fenestra rim.

As a result of these findings it became obvious that: *1.* the bony fenestra edge or rim can heal either by osteogenesis which obliterates the fenestra or by invagination of the inner surface of the flap into the fenestra gap and its adhesion to the bony edge of fenestra rim, which prevents osteogenesis and permits fenestra to remain patent; *2.* more windows stayed permanently open with the use of the fenestra nov-ovalis technique because the much wider fenestra created with this technique favored accidental invagination of the flap into the fenestra and its adhesion to the fenestra rim much more frequently than the previously made much narrower fenestra; *3.* osteogenesis as a rule did not take place extramarginal to fenestra rim because adhesion of the flap to the bony labyrinthine capsule outside the fistula prevented it.

Invagination and adhesion of the flap to the bony fenestra rim is undoubtedly the scientifically correct and logical solution for the prevention of osteogenesis of the fenestra rim. This should be obvious to any otologist of experience who could not have possibly failed to observe that the permanent

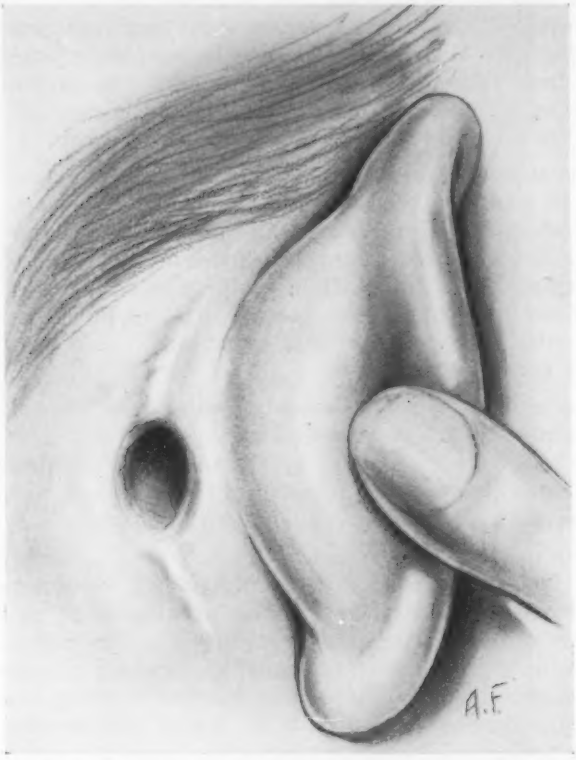


Fig. 5. Invagination and adhesion of postauricular skin and periosteum to bony edges of severed outer mastoid cortex as seen in every postauricular fistula.

postauricular fistula following the postauricular mastoidectomy is also the result of invagination of postauricular skin into the mastoid cavity and adhesion of its inner periosteal

surface to the bony edges of the severed outer mastoid cortex. When the postauricular skin and periosteum following mas-

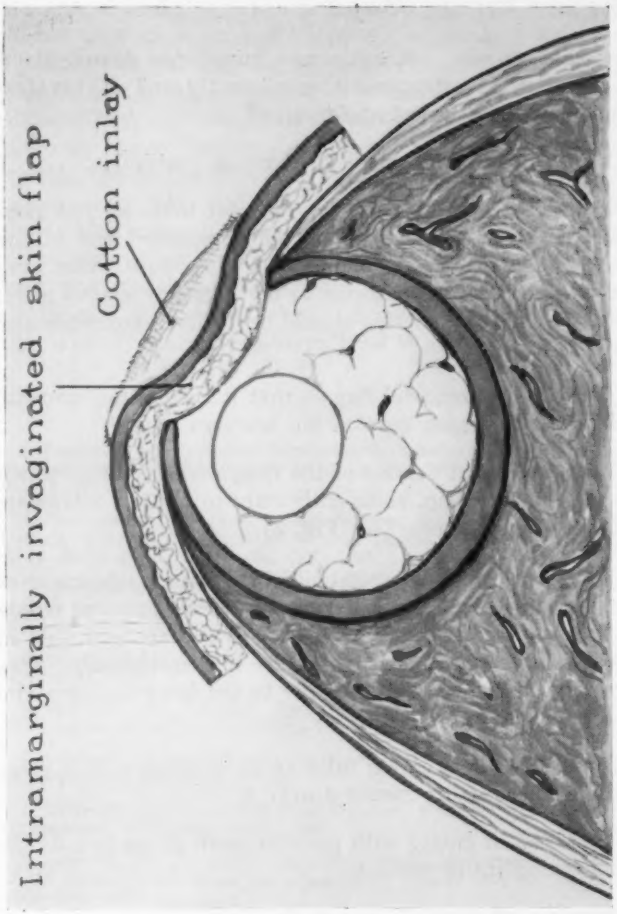


Fig. 6. Cross-section diagram demonstrating how to accomplish invagination of the tympanomeatal flap into the fenestra gap and the adhesion of its inner surface intramarginally to the fenestra rim for the prevention of osteogenetic closure of the fenestra nov-ovalis.

toidectomy healed by primary union the outer mastoid cortex as a rule regenerated underneath it and fistula was thus prevented. When the postauricular skin failed to heal by primary

union as a result of postoperative infection it often became invaginated and adherent to the bony edges of the mastoid cortex and a permanent postauricular fistula resulted (see Fig. 5).

How can we now, with this new knowledge gained about the prevention of osteogenesis, consistently and deliberately create a permanently patent fenestra?

1. Make fenestra on surgical dome of vestibule.

2. Make it as wide as possible. In order to do so, you must remove the anterolateral wall of the ampullated end of the bony external semicircular canal, thus exposing a wider area of the perilymph space anterior to the normally placed membranous labyrinth. Fenestra should be about 2 mm. wide and about 6 mm. long.

3. Place tympanomeatal flap so that it covers and contacts the entire bony region outside the fenestra gap.

4. Invaginate that portion of the tympanomeatal flap which faces the fenestra gap, sufficiently only to contact intramarginally the fenestra rim (see Fig. 6).

5. Place a cotton inlay moistened in Ringer's solution over the invaginated portion of the tympanomeatal flap and mould it so that it forces the inner surface to contact and stay in contact with the fenestra rim edges intramarginally. This favors its adhesion intramarginally to the bony fenestra rim (see Fig. 6).

6. Secure and hold cotton inlay in its position with a piece of paraffin mesh gauze moulded over it.

7. Pack rest of cavity with paraffin mesh gauze to hold the initial packing in its position.

8. Remove all packing except the first piece on sixth or seventh day.

9. Remove last piece on the tenth to twelfth day.

CONCLUSION.

I have finally observed and learned that invagination of the tympanomeatal flap into the fenestra gap and intramarginal adhesion of its inner surface to the relatively avascular bony fenestra rim prevents osteogenetic closure of the newly created fenestra. As a result of this observation it also became obvious that the reason osteogenesis did not as a rule take place in the relatively avascular bony labyrinthine capsule outside the fistula following fenestration surgery even when osteogenesis took place within fenestra rim was because the flap which became adherent to it prevented it. With this knowledge at hand we should now be able deliberately to prevent postoperative osteogenetic repair of the fenestra nov-ovalis and as a result obtain a much larger percentage of permanently patent fenestras than ever before.

REFERENCES.

1. LEMPERT, J.: Endaural Fenestration of External Semicircular Canal for Restoration of Hearing in Cases of Otosclerosis. *Arch. Otolaryngol.*, 31:711-779, May, 1940.

2. LEMPERT, J.; MELTZER, P. E.; SPECTOR, B.: Bone Repair Following Defects Made in the Skull of Cats by Means of Different Instruments. *Arch. Otolaryngol.*, 36:473-490, Oct., 1942.

3. LEMPERT, J.; MELTZER, P. E.; SCHALL, L. A., and WOLFF, D.: Osteogenesis Following Fenestration of the Vestibular Labyrinth of the Rhesus Monkey. *Arch. Otolaryngol.*, 46:512-527, Oct., 1947.

4. LEMPERT, J.: Fenestra Nov-Ovalis. *Arch. Otolaryngol.*, 34:880-912, Nov., 1941.

5. LEMPERT, J.: Lempert Fenestra Nov-Ovalis with Mobile Stopple. *Arch. Otolaryngol.*, 41:1-41, Jan., 1945.

6. LEMPERT, J.: Improvement of Hearing in Cases of Otosclerosis. *Arch. Otolaryngol.*, 28:42-97, July, 1938.

7. LEMPERT, J.: Bone-Dust-Free Lempert Fenestra Nov-Ovalis. *Arch. Otolaryngol.*, 47:280-288, Mar., 1948.

8. LEMPERT, J.: An Analytical Survey of the Evolutionary Development of the Fenestration Operation. *Ann. Otol., Rhinol. and Laryngol.*, 59:988-1019, Dec., 1950.

HISTOPLASMOSIS IN THE ADULT: CASE REPORT AND REVIEW OF THE LITERATURE.*†

DEAN M. LIERLE, M.D., and T. LYLE CARR, M.D.,
Iowa City, Iowa.

Histoplasmosis has been defined by Worgan[1] as a fungus disease involving the reticuloendothelial systems. It is of particular interest to otolaryngologists because of the ulcerative lesions of the mucous membranes of the upper respiratory passage and alimentary canal. Frequently the cervical vertebrae, frontal and maxillary bones and the middle ear tract have been affected. We will summarize briefly the findings of various observers and then report one of the four cases which have been recognized at the University Hospital during the last two years.

Histoplasmosis was discovered by Darling,[2] in 1905, while looking for evidence of kala-azar in smears of the spleen, liver and bone marrow. In 1906, he reported finding enormous numbers of small bodies, generally oval or round, most of which were within the alveolar epithelial cells; however, some appeared to be free in the plasma of the spleen and rib marrow. Darling thought the disease was caused by protozoan parasites. Twenty years elapsed before the next case was reported by Watson and Riley. According to Worgan,[1] DeMonbreun proved that the causative agent was not a protozoan parasite but a fungus which was called histoplasmosis capsulatum. It existed in two forms: one, a yeast-like form which grows in the reticuloendothelial system of man, and the other, mycelial, growing outside of the body.

*Read at the meeting of the Southern Section, American Laryngological, Rhinological and Otological Soc., Inc., Miami Beach, Fla., Jan. 17, 1951.

†From the Department of Otolaryngology and Oral Surgery and the Department of Internal Medicine, State University of Iowa, Iowa City, Iowa.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 15, 1951.

Worgan[1] reported that DeMonbreun, in 1933, isolated both the yeast-like and mycelial forms of the disease. He obtained a pure culture of the fungus which grew like "small white cotton tufts," distributed over the surface of dextrose and Sabouraud's agar. Although there were numerous colonies, on inoculation the mycelium has no aerial hyphae. He was unable to find the yeast-like forms in the original culture but later did find them. He described the organisms as oval bodies measuring about 3 microns in diameter, the cell being surrounded by a thick membrane containing one or more fat droplets and usually a single protoplasmic granule. Supravital staining with methylene blue revealed deeply stained granules. Each cell contained one or more vacuoles which took up the stain. If the yeast-like forms were allowed to lose their moisture, the mycelial form soon made its appearance.

The portal of entry of the fungus is not known. Some authors, workers, persons and students think that the mouth is probably the most frequent site of entrance because of the ulcerations of the mucous membrane of the mouth, pharynx and gastrointestinal tract. Others feel that the respiratory tract is the pathway of entrance because the pharynx, larynx and lungs often have lesions.

Hodgson, Weed and Claggett[3] made a survey of the cases reported up to 1949 and found 123 cases. They learned that the disease was rather widespread. Cases were reported from 25 states and 17 foreign countries; three-fourths were from the United States. Leopold[4] reported that in studies made by Gass (1943) and Lumsden (1939) a much higher figure of pulmonary calcification was found in nonreactors to tuberculin residing in the area of the eastern slope of the Mississippi River basin and the adjoining states and the western slope south of Iowa than in the rest of the country. The ages of the patients varied between seven and one-half weeks to 77 years, and about one-sixth were less than one year of age.

The disease appears to be more prevalent in males than females. Parson and Zarafonetis[5] reported histoplasmosis

occurring in a ratio of 51 males and 14 females. It is more prevalent in the white than the Negro race. Up to the present time histoplasmosis has not been found in the Arctic regions. It occurs equally in urban and rural areas. The larger number of cases now being reported indicates that the disease is not as rare as formerly thought.

The symptoms and signs vary in individual cases. Fever may be absent but is usually low grade, ranging from 100 to 102° F. but it may go as high as 107° F. Night sweats, malaise, anorexia, weakness and loss of weight occur during some stages of the disease in most instances. Cough, earache, sore throat, odynphagia, dysphagia, hoarseness, sputum, dysphonia, diarrhea, hemorrhage per rectum, arthritis and chills have been reported in many case histories. There may be localized or general lymphadenopathy and enlargement of the spleen and liver. Ulcerations of the mucous membrane of the tongue, mouth, pharynx and larynx are extremely painful. There were no consistent abnormalities in the urine, but in certain cases albumin, casts, red and white blood cells were found.

Parsons and Zarafonetis[5] report that the hemoglobin was reduced in proportion to the reduction in the number of red cells, but the anemia described in most instances has been hypochromic, either microcytic or normocytic in type. They also reported that the white count was consistently in the leucopenic range in 18 cases, normal in 11 cases, and consistently above 10,000 in nine cases. In their series, the differential counts showed no consistent changes and were subject to wide variations.

The majority of observers reported that in about half of the cases the lungs were affected. Ulcerative lesions of the mucous membrane of the pharynx, larynx and gastrointestinal tract occurred in about a third of the cases. Approximately 10 per cent had lesions of the skin. The sites most frequently involved are the lungs, spleen, liver, bone marrow, lymph nodes, gastrointestinal tract, adrenals, oral mucosa, kidney and larynx. Some authors have reported involvement of the middle ear, prostate and thyroid glands.

Grossly the typical pulmonary lesions have a soft yellowish white foci which resemble tubercules.[1] The average lesion measures from 5 to 15 mm. and is surrounded by a translucent fibrous capsule. Worgan[1] at autopsy found the parasite to be a pear-shaped crescentic organism, two to four microns in size and surrounded by a thick refractile capsule. They all contained one or more nonstaining vacuoles and one or more dark chromatin masses. Usually there was an invasion of the tissues by mononuclear cells which contained many of the parasites and red blood cells.

DISTRIBUTION OF PATHOLOGIC LESIONS.

Dean[6] reported a case with involvement of the larynx in which the epiglottis was thick, nodular and pale, and the whole interior of the larynx appeared to be white and nodular. He also stated that the false and true cords were so extensively involved that they appeared to be entirely obliterated. The glottic chink was converted into an irregularly rounded tube. The organism was recovered from the granulations about the tracheotomy wound.

Duncan[7] also described a case reported by Miller, Johnstone and Bostick, in which there was an ulcerating nodule about 1.5 cm. in diameter on the dorsal aspect of the tongue. The diagnosis was made by scraping the ulcer and isolating the fungus. There was marked lymphadenopathy, and enlargement of the liver but not the spleen.

Parson and Zarafonetis[5] made a splendid report of the distribution of the lesions in their cases. A summary of their report was as follows: Of 61 cases, 19 had some skin lesions present; nine had ulcers; four had papular eruptions; six had purpura occurring in the last two or three days of life. Petechial hemorrhages and bullous lesions were found once. The ulcers frequently were deep with sharp, steep edges, having a punched out appearance. In one case of involvement of the tongue there was considerable edema around the border of the lesion. They also reported involvement of the middle and external ear canal in eight cases and a true otitis media in five cases. Organisms were found in the pus in one case of

middle ear infection. Ulcers of the mucous membrane of the mouth most frequently involving the tongue were found in 14 cases; four other cases had ulcers of the mucous membrane of the nose and mouth. Perforation of the nasal septum was noted in three of their cases. The perforations of two were known to have been produced by infection with histoplasmosis capsulatum. There were granulomatous and ulcerative lesions of the pharynx and larynx in 10 cases. Superficial lymph nodes were discovered in 29 of the 61 cases with enlargement in 16 cases. Cervical nodes alone were affected in five cases, and in conjunction with other regional nodes in four other instances.

Agress and Gray[8] reported histoplasmosis in a boy seven months of age with a saddleback deformity of the nose, perforation of the septum, mucopurulent rhinitis, ulcers of the buccal mucosa and lesions about the mouth suggestive of rhagades. The liver and spleen were enlarged, but there was no lymphadenopathy. The Kahn and Wassermann tests were negative.

METHOD OF DIAGNOSIS.

The diagnosis of histoplasmosis can be established only when the clinician suspects it and includes it as a possibility even in the absence of skin and mucous membrane lesions. Diagnosis by biopsy has been the most successful of all methods during life. Sternal marrow aspiration often enables the examiner to identify the organisms within phagocytic cells of reticuloendothelial origin, and culture of this aspirated material makes this an even more reliable procedure. Blood smears may prove a source of recognizable histoplasma capsulatum in the generalized type but thick film preparations increase the probability of success. Blood cultures likewise help. In the same regard, culturing of biopsy material obtained from ulcerated involved areas may aid in establishing the diagnosis. If a recent or old infection is suspected, one may use an intradermal injection from a filtrate of cultured medium in which the fungus has grown. A positive test is indicated by erythema and a wheal at the site of the injection.

TREATMENT.

Treatment with pentnucleotide, fuadin, irradiation, neostam, sulfonamides, sodium thiosulfate, iodides, ionized metals, potassium tartrate, neoarsphenamine, acetarsone, autogenous vaccines, blood transfusions and various other drugs bears witness to the lack of a specific cure. Histoplasmosis is almost universally fatal, usually within a few months and rarely some years after the onset of the disease. Parson and Zarafonetis[5] reported that in most cases clinical signs of infection lasted less than one year, the average in 39 cases being about five months. In one group, however, there were four cases that lasted four, eight, 10 and 16 years, respectively.

CASE REPORT.

Case No. 50-8604: A 34-year-old farmer was well until Sept. 17, 1949, when he developed a dry cough, chills, sweats, weakness, and a temperature of 103.01°. He was unable to work. In October, 1949, a palpable spleen and temperature of 106° were noted.

A sore throat first occurred in January, 1950, when biopsy of a laryngeal lesion revealed no malignancy. In March, he consulted a neighboring clinic. A biopsy of the larynx was obtained and the tissue section demonstrated histoplasma capsulatum. Within a few days after returning home, he was unable to talk and had progressive difficulty in swallowing food. He complained of a sore throat with pain radiating to both ears. He was unable to swallow even saliva. Diplopia occurred on July 7, 1950; and 10 days later there was sudden loss of function of the right arm and leg without unconsciousness. A few days later dyspnea began with rapid aspirations. He had a chill and became disoriented. He had received alone or in combination numerous antibiotic and sulfonamide drugs with progression downward. On admission, there was emaciation, dehydration, mental confusion, aphonia, fever and rapid shallow respiration. Hypertension of 170/100 was present and continued throughout the illness. Right external rectus muscular paralysis and a droop of the right face excluding the forehead were evident. Two eroding ulcerative lesions were on the tongue. A well healed thyroidectomy scar was noted. The lungs contained many moist rales especially on the right. Complete flaccid paralysis of the right arm and right leg were present.

The appearance of the larynx in our case was very similar to the one reported by Dean.[6] It was generally pale and edematous, and one could not distinguish between the false and true cords, the normal landmarks being obscured. The larynx was tubular in appearance with some obstruction to the airway. The arytenoids appeared hypertrophic and edematous. There was an irregular, cauliflower, pale mass about 1.5 by 1 cm. in size involving the intra-arytenoid space, and extended into the chink of the glottis. The patient was having some difficulty in swallowing and an esophagoscopy was attempted, but we were unable to pass the scope because of a definite stricture at the level of the cricopharyngeus. The mucous membrane was pale and edematous, and it was impossible to pass a 2 mm. feeding tube.

Gastrostomy was performed and in a few days all necessary fluids plus a more than adequate diet were being given.

There was a hypochromic anemia, uremia, hypoproteinemia, albuminuria, and continuing temperature elevation with no marked change from the previously described critical picture. Bronchoscopy was necessary on several occasions, as well as almost constant pharyngeal suction.

Direct examination and culture of the blood and bone marrow failed to reveal any organisms. Fungus was cultured from the tissue biopsy on several occasions but at no time would it produce tuberculated chlamydospores and, therefore, could not be positively identified as histoplasma capsulatum. A tissue section taken from the larynx in March, 1950, and sent to us by Dr. L. B. Woolner was positive for histoplasma capsulatum.

In addition to the usual supportive and other therapeutic measures, we attempted the administration of ethyl vanillate, a fungicidal agent which shows considerable promise in generalized histoplasmosis infection in children.[9] We were unable to obtain satisfactory blood levels, however, and death occurred seven weeks after admission. Autopsy permission was refused.

We have attempted to call attention to this infection whether generalized or localized. We feel that it is of special interest to the otolaryngologist in view of the fact that many of the lesions involve the mucous membrane of the upper respiratory and digestive tracts, sinuses, external ear and middle ear.

We thank Dr. L. B. Woolner, of the Mayo Clinic, for the use of the tissue section, and Dr. A. P. McKee, of the Department of Bacteriology, College of Medicine, State University of Iowa, for his work in culturing and identifying the fungus.

BIBLIOGRAPHY.

1. Worgan, David K.: Histoplasmosis: A Summary of Known Facts About the Disease. *Bull. School of Med., Univ. Md.*, 30:29, Oct., 1945.

2. Darling, S. T.: A Protozoan General Infection Producing Pseudotubercules in the Lungs and Focal Necroses in the Liver, Spleen and Lymph Nodes. *Jour. A. M. A.*, 46:1283, 1906.

3. Hodgson, Corrin; Weed, Lyle Al., and Claggett, O. T.: Pulmonary Histoplasmosis: Review of Published Cases and Report of an Unusual Case. *Jour. Thoracic Surg.*, 20:97, July, 1950.

4. Leopald, Simon S.: Respiratory Diseases. The Cyclopedia of Med., 351, 1946.

5. Parsons, Robert J., and Zarafonetis, C. J. D.: Histoplasmosis in Man. *Arch. Int. Med.*, 74:1, 1945.

6. Dean, L. W., Jr.: Histoplasmosis of the Larynx. *Arch. Otol.*, 36:390, 1942.

7. MILLER, H. E.; KEDDIE, FRANCES M.; JOHNSTONE, J. G., and BOSTICK, W. L.: Histoplasmosis Cutaneous and Mucomembranous Lesions, Mycologic and Pathologic Observations. *Arch. Dermat. and Syph.*, 56:6:715-737, Dec., 1947.

8. AGRESS, HARRY, and GRAY, S. H.: Histoplasmosis and Reticuloendothelial Hyperplasia. *Amer. Jour. Dis. Child.*, 57:573, 1939.

9. CHRISTIE, AMOS; MIDDLETON, JAMES G.; PETERSON, J. CYRIL, and McVICKOR, DAVID L.: Treatment of Disseminated Histoplasmosis with Ethyl Vanillate. In press.

BRONCHIAL ADENOMA.*

CHEVALIER L. JACKSON, M.D., and CHARLES M. NORRIS, M.D.,
Philadelphia, Pa.

Bronchial adenoma is one of the most important and most interesting tumors found in the bronchi. The histopathologic diagnosis is difficult, and these lesions are often mistaken for carcinoma. It is important to discover them early, because if not treated they produce bronchial obstruction of marked degree, and subsequently bronchiectasis, abscess and empyema; furthermore, they *must be* differentiated from carcinoma because they require less radical treatment.

The rôle of bronchoscopy is especially important in the diagnosis and treatment of these tumors because they almost always arise in the larger bronchi. Certainly everyone admits that they can generally be seen and biopsied by bronchoscopy, regardless of the efficacy of bronchoscopy in treatment.

Our experience with these tumors has been presented in previous articles: Jackson and Konzelmann,[1,2] Jackson[3,4] (in German and Italian), Jackson, Konzelmann and Norris,[5] and Jackson and Norris.[6] In the last of these previous papers, given before the annual meeting of the American College of Chest Physicians in San Francisco, Calif., last June, our previous papers were summarized, and 40 cases were reported. Seventeen of those cases had never been reported before, and the remaining cases those previously reported, and presented again with late follow-up.

Symptomatology and Diagnosis. The symptomatology of bronchial adenoma has already been extensively discussed,

*Read at the meeting of the Southern Section, American Laryngological, Rhinological and Otological Society, Inc., Miami Beach, Fla., Jan. 17, 1951.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 15, 1951.

and it is well known that this type of tumor produces *hemoptysis* early, is generally accompanied by *cough*, more often productive than not, and above all by the physical and X-ray signs of *bronchial obstruction*. The *diagnosis* is made on the basis of these findings, confirmed by the bronchoscopic appearances, and the opinion of the pathologist based upon study of specimens removed for biopsy. Cytologic studies of secretions have not been so helpful as in carcinoma, but by the nature of the lesion, actual tissue specimens can generally be obtained, because the tumors generally arise in the accessible portions of the bronchial tree.

Pathology. It has always seemed to us that the crux of the confusion concerning these tumors lay in the histopathology. The typical histopathologic picture has been described by Konzelmann.[1] The subdivision into different types, as for example "*carcinoid*" and "*cylindroma*" types as suggested by Hamperl[7] is useful. There has been much discussion as to whether bronchial adenomas should be considered benign or malignant, but we believe that they should be considered "borderline" tumors[8] for the present. Malignancy ordinarily depends chiefly on tendency to local invasion, and ability to metastasize. Certainly the appearance of local invasion is given by these tumors, as was noted in the early study of the subject by Wessler and Rabin.[9] These authors pointed out, in 1932, that it was characteristic of bronchial adenoma to extend between the bronchial cartilages and for some of the tumor to be extrabronchial; however, we believe that this in itself cannot be considered a malignant characteristic, because it is due simply to the fact that the tumor arises from structures which occur between the cartilages rather than on the mucosal surface. Neither can involvement of contiguous lymph nodes be considered final proof of malignant character. Many of the cases of distant metastasis, as for example to the liver, have been proven to be cases of erroneous diagnosis, but it may be true that a few of the cases of liver metastasis which have been reported have represented true metastasis of a bronchial adenoma. We admit this, though such metastasis has not been noted in any of our own cases.

Treatment. We believe that irradiation has been of no proven value; that in many cases bronchoscopic removal is curative, and that in many other cases it constitutes efficacious preoperative or palliative treatment; that bronchotomy as shown by Goldman[10] and some others offers a good prospect of cure by a conservative surgical procedure in suitable cases; and that surgical resection by lobectomy or pneumonectomy is definitely indicated in many other cases, because of impossibility of complete local removal, compression due to persisting extrabronchial tumor; or irreversible changes in the distal portion of the lung. Finally, we very definitely believe that in dealing with bronchial adenoma, cases should be individualized in selection of treatment method, and resection should not be resorted to automatically as in carcinoma.

Clinical Experience. As mentioned above, our series of reported cases now includes 40 patients; 36 of these are considered to be of the carcinoid type, and four of the cylindroma type. Of these 40 cases, 26 patients were treated by bronchoscopic measures only, eight by lobectomy (with one postoperative death), and three by pneumonectomy (with one postoperative death); two patients had biopsy only and were lost track of. Of the five patients in the entire series of 40 who have died, two died postoperatively, though neither of the postoperative deaths occurred at the Temple University Hospital. All nine of the patients operated upon by Dr. W. Emory Barnett and Dr. George Rosemond at the Temple University Hospital (seven lobectomies and two pneumonectomies) survived. Two of the patients who died of disease were typical cylindromas and were treated bronchoscopically because the tumors were too close to the carina to permit pneumonectomy, though they would have been subjected to pneumonectomy if this had not been the case. The other two patients with cylindroma are living and symptom-free, apparently quite well under control by bronchoscopic treatment at long intervals.

Summing up, of the 40 patients in the series, five have died, two of them postoperatively; two were lost track of, and 33 are living and well, though some require occasional treatment and all return for occasional check-up examination.

REFERENCES.

1. JACKSON, CHEVALIER L., and KONZELMANN, FRANK W.: Bronchoscopic Aspects of Bronchial Tumors with Special Reference to So-Called Bronchial Adenoma. *Jour. Thoracic Surg.*, 6:312, Feb., 1937.

2. JACKSON, CHEVALIER L., and KONZELMANN, FRANK W.: So-Called Adenoma of the Bronchus. *Ann. Otol., Rhinol. and Laryngol.*, 50:1264, Dec., 1941.

3. JACKSON, CHEVALIER L.: The Bronchoscopic Diagnosis and Treatment of Bronchial Tumors. *Ztschr. f. Hals-, Nasen- u. Ohrenhil.*, Band 40, 4/5 Heft, 1936.

4. JACKSON, CHEVALIER L.: Adenoma Bronchiale. II Valsala, 1937.

5. JACKSON, CHEVALIER; KONZELMANN, FRANK W., and NORRIS, CHARLES M.: Bronchial Adenoma. *Jour. Thoracic Surg.*, 14:98, Apr., 1945.

6. JACKSON, CHEVALIER L., and NORRIS, CHARLES M.: The Rôle of Bronchoscopy in the Treatment of Bronchial Adenoma. *Dis. Chest* (in press).

7. HAMPERL, H.: Uber Gutarige Bronchial Tumoren (Cylindroma und Carcinoide). *Virch. Arch. f. Path. Anat.*, 300:46-88, 1937.

8. JACKSON, CHEVALIER, and JACKSON, CHEVALIER L.: Bronchoesophagology. Philadelphia, Pa.: W. B. Saunders Co., 1950.

9. WESSLER, R., and RUBIN, C. B.: Benign Tumors of the Bronchus. *Amer. Jour. Med. Sci.*, 183:164-180, 1932.

10. GOLDMAN, ALFRED: Malignant Nature of Bronchial Adenoma. *Jour. Thoracic Surg.*, 18:137-148, Apr., 1949. Also, GOLDMAN, ALFRED, and CONNER, CHARLES: Benign Tumors. *Dis. Chest*, 17:644-680, June, 1950.

ANTRO-ALVEOLAR FISTULAE.*

FREDERICK T. HILL, M.D.,†
Waterville, Me.

While the majority of antro-alveolar fistulae follow dental extractions, they may result from infection, cysts, or tumors of the upper jaw, trauma, or as a complication of operations on the antrum. Successful management depends upon careful study of the individual case and the application of a procedure calculated best to meet the existing conditions. This may vary from simple approximation of tissue to the use of fairly extensive plastic methods. Control of antral infection is essential. Treatment must be based upon sound anatomic and physiologic principles.

The importance of cooperation between the dentist, the oral surgeon, and the rhinolaryngologist cannot be overemphasized, particularly in the field of prevention.[1-3] The potential dangers of inadvertently entering the antrum in the extraction of teeth in close relation to the floor, and of subsequent sinus infection should be kept in mind. Should extraction of such a tooth be indicated, the dentist could well assure himself that antral infection was not already present. In the case of already existing sinusitis, measures to control this, such as intranasal antrostomy prior to extraction, would avoid many of these complications. The dentist and the oral surgeon should realize that infection of the antrum is a rhinological problem and any involvement should be managed by one trained in rhinology.

In large antra with thin walls the roots of the bicuspid

*Read at the meeting of the Eastern Section, American Laryngological, Rhinological and Otological Society, Inc., Philadelphia, Pa., Jan. 12, 1951.

†Thayer Hospital, Waterville, Me.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 15, 1951.

teeth and the first, second, and sometimes the third molars, or even the canines, may mound up in the floor of the sinus. Sometimes the bone may be missing with only the mucoperiosteum separating the roots from the antrum.

Fistulae of dental origin may occur as a result of rough attempts at extraction, the breaking off of roots and pushing them upward into the antrum, too strenuous curettage of sockets, interlocking roots sometimes looped over a thin antral

Fig. 1. Showing modified canine incision extending downward to include outer margin of fistula.

floor, or roots with marked hypercementosis. Fusion of molars and bicuspids may be a cause of fistula following extractions. In a case of the author's, the dentist in attempting extraction of the first molar had removed the first, second, and third molars, together with surrounding process, all in one

mass. Most of these fistulae could be prevented by a more careful technique or previous Roentgen study on the part of the exodontist. Approaching a retained root from the side, by removal of some of the lateral plate, and manipulating it downward from above, generally will avoid pushing the root into the antrum. Any granulomatous tissue in the socket should be carefully removed, avoiding the forcible use of

Fig. 2. Soft parts elevated exposing fistula. This drawing was made from a case with multiple fistulae. One in the site of the second molar and a smaller one in the socket of the canine.

sharp curettes which might result in pushing through the thin floor into the antrum. Study of the dental Roentgenograms should allow the exodontist to plan his procedure so as to avoid undue trauma to the antral floor. Sometimes splitting the crown of a molar will allow removal in the plane of the long axis of distorted roots.

When a root or, as sometimes occurs, an entire tooth has been forced into the antrum, it is preferable to remove this by opening the antrum through the canine fossa, although

occasionally it is possible to effect removal through an intranasal antrostomy. Efforts to manipulate any foreign body down through the fistula usually adds to the trauma and makes healing more difficult.

While rarely seen, to be sure, the possibility of neoplastic changes occurring in an antro-alveolar fistula should be kept in mind.

One patient in our series,[4] a man of 41, was seen because of pain and swelling in the upper jaw and neck of one month's

Fig. 3. Necrotic bone surrounding fistula removed exposing interior of antrum. Reversed or hinged flap from posterior tuberosity outlined.

duration. He had had a discharging antro-alveolar fistula following the extraction of teeth 20 years before. Examination revealed a large ulcerative tumor mass in the alveolar process with a fistulous tract extending into the antrum. The submaxillary area was greatly swollen with a mass of

broken-down metastatic nodes. Biopsy was reported as epidermoid carcinoma. Extensive electrocoagulation of the primary lesion, followed by irradiation, using radium as well as external Roentgen therapy resulted in temporary improvement; but attempts to eradicate the metastatic mass in the neck were unsuccessful, and the patient succumbed in eight months.

Many fistulae may heal spontaneously or with a modicum of intelligent treatment directed to the antrum, but a tract of any size, from the mouth with its rich and variegated bac-

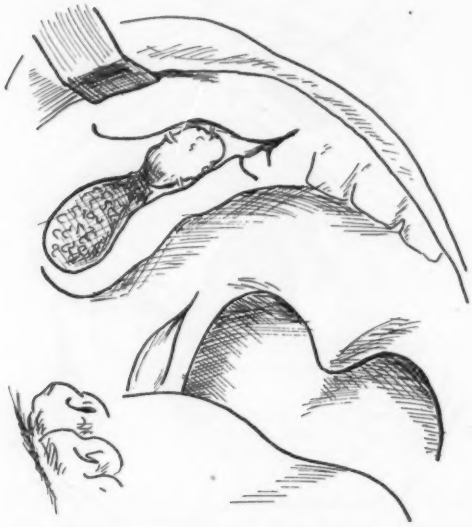


Fig. 4. Hinged flap turned forward covering fistula and presenting raw surface for sliding buccal-gingival and palatal flaps. Anchored with one or more catgut sutures.

terial flora to the antrum, is most liable to result in infection of the sinus, if indeed this is not already present, due to chronic sinusitis.

The advantages of early attention in these cases are obvious. This may result in spontaneous healing of the fistula or a

satisfactory response to simple methods of closure. Persistence of the condition not only increases the technical difficulties in achieving a closure but also exposes the patient to all of the annoyances and hazards of a chronic sinusitis.

Roentgenograms of the sinuses are mandatory. If there is little evidence of a pre-existing chronic sinusitis conservative

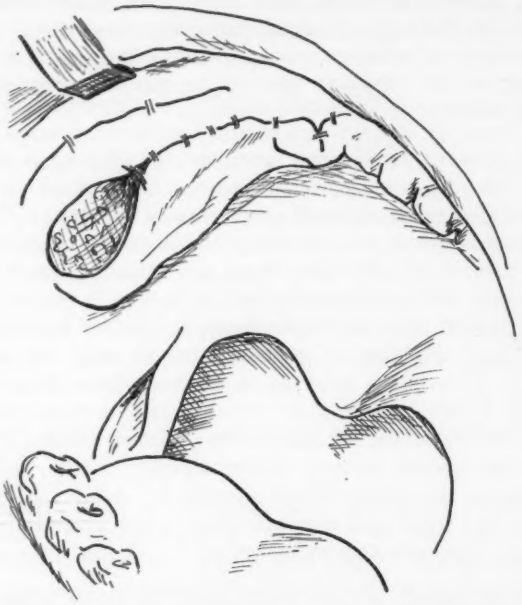


Fig. 5. Bipedicled buccal-gingival and palatal flaps brought together over raw surface of hinged flap and sutured with silk. Mucous membrane will grow across denuded area of posterior tuberosity.

measures may be employed. These include irrigation of the antrum, parenteral antibiotic therapy based upon culture of the organism, as well as intranasal use of vasoconstrictor sprays. Packing, irrigation, or any manipulation of the fistulous tract, including the application of caustics, is contraindicated; not only are these useless but oftentimes may add to the difficulty.

When the antral lining is markedly thickened, and there is evidence of pre-existing chronic sinusitis, an intranasal antrostomy is indicated. Little is to be gained by procrastination. Intranasal drainage should be established as early as possible. The removal of secretions by the suction cannula through the antrostomy opening is, in my opinion, preferable to lavage, although many operators employ irrigation. Some fistulae, if small and seen early, may close spontaneously following such therapy. Usually, however, closure will require the elevation of adjacent mucoperiosteal flaps, with sufficient lowering of the alveolar process to permit approximation without tension.

A number of techniques, such as the Dunning[1] and the Welty,[5] have been employed, based on the principle of covering the defect with adjacent mucosa from the palate. The use of the saddle-plate, as described by Dunning, to protect the suture line, is helpful when there are adjacent teeth for its attachment. Ashley[6] described a procedure in which a long mucoperiosteal flap was taken from the palate and the anterior portion, denuded of mucosa, used to plug the opening. Isografts of cartilage inserted in the tract have been used to close the fistula. McQuiston[7] employs sliding flaps from the palate and the buccal surface of the alveolus, about 1.5 cm. in width, the buccal incision being similar to the usual one employed in the Caldwell-Luc operation. After curettage of the fistulous tract and removal of infected granulatious tissue, these flaps are approximated with silk mattress sutures.

Local anesthesia is very satisfactory in all of these procedures. Good oral hygiene is essential. Any dental caries should be eliminated before attempting any plastic procedure on the alveolar process.

The fistula of long duration will have developed a tract lined with mucous membrane which will require removal. The antrum is always infected and usually should be approached through the canine fossa using the Caldwell-Luc technique. Occasionally one may encounter a small fistula of this chronic type, in which the "folding-door" type of incision,[8] followed

by undermining the lining of the tract with a narrow cataract knife and turning this cored tract inward, may prove successful. The flaps of the "folding-door" are then closed by mattress sutures of deknatel. A slight amount of the alveolar process may be removed with ronguers to facilitate approximation. Sutures are removed in four or five days.

In all of these procedures patients are cautioned against sucking on the operated area, manipulating it with the tongue, or forcible blowing of the nose.

The management of a large fistula of long duration may prove rather complicated and resist simpler methods of closure. In dealing with this type the Caldwell-Luc operation[9] should be combined with the plastic procedure on the fistula. Formerly it was my custom to do this in two stages, the usual Caldwell-Luc operation being followed in a week or 10 days by the plastic procedure on the fistula. This had the disadvantage of making impossible a strong buccogingival flap, this being limited by the horizontal canine fossa incision. For some years I have employed the one-stage operation, modified as follows for these large fistulae.

The indicated antibiotic therapy is given preoperatively and carried on postoperatively.

Local anesthesia is employed, after preliminary medication with nembutal (pentobarbital sodium), gr. 1ss, one and two hours before operation. The lateral wall of the inferior meatus of the nose is anesthetized by applying a thin strip of cotton moistened with cocaine 10 per cent and adrenlin chloride 1-1000. Two to three cc. of procaine 2 per cent are then injected slowly into the infraorbital foramen. This may be done directly through the cheek after the foramen is located by palpation. The posterior palatine foramen is then injected, followed by infiltration in the anterior part of the hard palate and along the buccogingival groove.

A modification of the usual canine fossa incision is employed, curving the mesial portion downward to include the lateral margin of the fistula. This allows for a more substan-

tial buccal flap than is possible with the usual horizontal incision and also avoids the scar tissue inevitable in the two-stage procedure. A sharp submucous dissector is used to elevate the periosteum until the wall of the antrum is sufficiently exposed. The defect in the bone is generally lateral, as most teeth are extracted laterally. This allows adequate exposure of that area, so that any necrotic bone may be removed with ronguers. Usually this gives an opening adequate to remove the infected antral lining and make the intranasal window.

After the antral part of the operation is completed a hinged or reverse flap of mucous membrane is dissected from the alveolar process, usually posterior to the fistula, and turned forward to close the weak posterior angle and provide a raw surface for sliding flaps of buccogingival and palatal mucous membrane. The apex of this flap is anchored to the lower or unelevated margin of the original incision just anterior to the fistula with a No. 0 catgut submucous suture. An incision is now made in the buccal mucous membrane, somewhat parallel to the original incision, forming a ribbon, or sliding flap, approximately 2 cm. in width, by dissecting with scissors submucously and leaving both ends intact. This buccogingival flap should be fairly generous, care being exercised to avoid Stenson's duct. With this bipedicled flap, with both ends intact and only its midportion sliding downward, adequate blood supply is possible. It has proven quite as satisfactory as the delayed flap.

The palatal flap, also bipedicled, is cut about midway between the midline of the palate and the alveolar process and stops short of, or swings lateral to, the posterior palatine foramen. Both flaps are so freed that their central parts may be approximated without tension over the hinged or reverse flap, using single or mattress deknatel sutures. If projecting process prevents this it may be lowered. Usually in the large fistula only the palatal plate of the process will require trimming.

The buccal incision may be loosely approximated with one or two sutures. The tissue in this region is so free and pliable

that it heals readily. It is advisable, however, to caution the patient to avoid, as much as possible, motion of that side of his face, or pushing the tongue towards the cheek. The palatal incision is not sutured and usually heals readily and with much less discomfort for the patients than with the median palatal incision.

This procedure allows for adequate exposure of all diseased areas and provides a bed for the mucous membrane flaps, bringing raw surface against raw surface. It seems to meet the requirements of plastic surgery and, in our hands, has proven most satisfactory. It does not require a large palatal flap and can be used even in cases having a torus palatinus.

In the management of any antro-alveolar fistula of long duration it is essential to eradicate thoroughly any osteomyelitis. Sometimes this may require considerable removal of bone of the anterior antral wall. In the past when employing the two-stage operation, this procedure, at times, has resulted in an operative defect in the canine fossa, necessitating repair with a delayed flap.[10] In our experience this has not occurred since adopting the one-stage procedure as above outlined.

In endentulous patients it is important to preserve sufficient alveolar process to insure the satisfactory use of a denture. The tuberosity of the maxilla may be considered as the posterior buttress, very necessary for an upper denture. If this is preserved a denture usually can be made to function satisfactorily. In this connection it is well to remember that all patients may be candidates for full dentures at some later time.

AFTER-TREATMENT.

Patients are allowed out of bed the day following operation. Soft diet is given, but they are instructed to chew on the unoperated side. Frequent mouth washes are prescribed, and any accumulated food is gently removed with cotton swabs. The incisions may be painted with methylene blue or gentian violet 1 per cent, once or twice daily. Sutures are removed on the fourth or fifth day. Any accumulated blood

or debris is removed from the antrum by cannula suction intranasally twice a day. Healing usually takes place by the seventh or eighth day, and if dentures are worn they may then be resumed.

In fistulae resulting from cysts or tumors the same general principles apply, although first consideration must be given to thorough eradication. This may result in very large defects either in the canine fossa or in the palate, so that the use of a prosthesis may be required. In cases of malignancy it is advisable to maintain adequate openings for the purpose of postoperative observation. In such cases flushing the cavity once or twice daily with potassium permanganate (1-10,000), or at times the insufflation of sulfanilamide powder, is helpful in overcoming any odor and keeping the cavity clean.

CONCLUSION.

The majority of antro-alveolar fistula are of dental origin and are preventable.

Successful management depends upon the control of antral infection, whether pre-existing or resulting from the fistula. The greatest hazard is gravity drainage.

The problem of management may be quite simple or extremely complicated. No one procedure is adaptable for all cases. Some may heal spontaneously or respond to conservative measures and simple methods of closure; others may require carefully planned plastic procedures.

A one-stage technique which has proven satisfactory in the most resistant type is presented.

REFERENCES.

1. Dunning, H. S.: Maxillary Sinus of Oral Origin. The Laryngoscope, 35:766, Oct., 1925.

2. Brown, L. E.: Relationship of Teeth to Pathology of the Maxillary Sinus. *Ann. Otol., Rhinol. and Laryngol.*, 34:150, Mar., 1925.

3. Lyons, H. R.: Treatment of Alveolar Fistula Secondary to the Extraction of Teeth. *Trans. Am. Laryngol., Rhinol. and Otol. Soc.*, 31:81, 1925.

4. HILL, F. T.: Carcinoma Occurring in an Antro-Alveolar Fistula. *Ann. Otol., Rhinol. and Laryngol.*, 56:4, Dec., 1950.

5. WELTY, C. F.: Closure of Fistulous Openings Through the Alveolar Process into the Antrum of Highmore. *Trans. Sec. Laryngol., Otol. and Rhinol., Am. Med. Assn.*, 1920, p. 198.

6. ASHLEY, R. E.: Method of Closing Antro-Alveolar Fistulae. *Trans. Am. Laryngol., Rhinol. and Otol. Soc.*, 45:440, 1939.

7. MCQUISTON, R. J.: Maxillary Sinusitis of Dental Origin and the Management of Antral Fistula. *Trans. Am. Laryngol., Rhinol. and Otol. Soc.*, 1945, p. 348.

8. HILL, F. T.: Management of the Alveolar Fistula. *Arch. Otolaryngol.*, 40:167:70, Sept., 1944.

9. HILL, F. T.: Management of Antro-Oral Fistulae. Grad. Lecture No. 331. Am. Acad. Ophth. and Otolaryngol., 1944.

10. NEW, G. B.: Use of the Delayed Flap in Secondary Operations on the Palate and Antrum. *Trans. Am. Laryngol., Rhinol. and Otol. Soc.*, 29:344, 1923.

EMOTIONAL FACTORS IN OTOSCLEROSIS.*

EDMUND PRINCE FOWLER, M.D.,
New York, N. Y.

Having set forth in several publications concepts underlying the effects of emotional disturbances upon the organ of hearing, and especially in respect to tinnitus, vertigo, and certain types of obstructive, neural and sudden deafness, it is now my purpose to call attention again to these in relation to otosclerosis; not only to examine them as concomitants or resultants of this disease, but also as possible factors in its etiology.

Were it not for our abysmal ignorance as to the cause of otosclerosis, and our utter helplessness in curative measures, I should hesitate to present the tenuous data I here set forth; however, even the faintest lead should not be neglected. Time and experimentation will prove whether it will develop into something worthwhile.

It is to be emphasized that up to the present hour otoclerosis has never been shown to result from any specific cause. It may develop, or cease to develop, in the presence or in the absence of other disease entities. It is markedly less prevalent in the Negro race, and less prevalent in males than in females, and as far as we know, in all races. It has never been produced or revealed in animals in a form surely recognizable as the same lesion. It is thought that its etiology may be hidden in some inherited factor.

My own observations lead me to believe that in addition to the psychic reactions observed in chronic deafness from any

*Read at the meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society, Inc., Philadelphia, Pa., Jan. 12, 1951.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 18, 1951.

cause, loss of hearing from otosclerotic ankylosis of the stapes footplate is accompanied by emotional behavior peculiar to itself. In the first place for some reason it as a rule engenders, or is associated with, more increasing concern, alarm, and frustration than the same amount of deafness from recurrent or chronic inflammatory otitis media, and whether or not the deafness is in a stationary stage, or progressing; also whether or not the threshold audiograms show like overall losses and patterns similar to those of other forms of impedance deafness. I believe that there is on the average a higher incidence of certain symptoms dependent upon emotional factors in otosclerosis than in progressive impedance deafness from other causes.

Among such signs and symptoms are: disturbing tinnitus, slight subjective unsteadiness, a tendency to ignore or deny the first indications of deafness, aversion to consulting an otologist, and even to resent suggestions that the hearing is not "perfectly all right," persistent objection to using a hearing aid or studying lipreading, until long after these are clearly indicated. Although many of these behavior patterns are observed in all types of moderately severe deafness, they are on the average more marked in otosclerosis. This may be in part because inflammatory or suppurative otitis is more understandable, a less mysterious lesion with a less insidious onset of deafness than otosclerosis, or it may be because the patient with otosclerosis is on the average more emotionally unstable, or as I prefer to put it, more emotionally sensitive. It cannot be because the hearing cannot be aided in otosclerosis, as it is well known that hearing aids are especially useful in overcoming the impedance deafness due to ankylosis of the stapes; in fact they are more useful than in other types of deafness.

In otosclerosis the hearing besides changing with atmospheric conditions, and all of the other factors within or without the body which affect it, also seems to be very sensitive to psychic states or episodes. After such episodes the hearing frequently appears to be permanently at a lower level than it was before, and if episodes are frequent or prolonged it not

infrequently is permanently lost to some degree. This suggests that such progressiveness in the deafness is caused by an activation of the lesion.

In otosclerosis it would seem that there is a less homeostatic, more hypersensitive or unstable autonomic neural balance which is, therefore, subject to frequent and more damaging nervous repercussions than in other forms of ear impedance disorders.

It is an interesting fact that certain types of neural deafness (even total so-called "nerve deafness") also may and frequently do follow emotional or psychic disturbances,[1] and appear to be inheritable. It is also a fact that highly unstable autonomic nervous systems are commonly inherited. They occur in some families more frequently than in others.

The question arises, — is otosclerosis or potential otosclerosis inherited as such, or rather as a result of the inheritance of a hypersensitive autonomic nervous system which is capable of acting on a tissue with a peculiar susceptibility to this particular form of bone dyscrasia? The idea is intriguing. Even severe disturbances in autonomic function need not be manifest by definite lesions in other parts of the body because of some anatomical, hormonal, neurovascular or metabolic disturbance, to be capable of causing disease in the unique bone of the capsule of the ear.

Pathophysiological disorders may be and often are precipitated by emotional disturbances in more or less characteristic and predisposed (hereditary) types; it is sometimes uncertain whether as cause or effect. A lesion produced by a neurovascular disorder may be apparent only long after the activating factor has faded into the background, so that there seems to be no causative relationship involved.

If people who acquire otosclerotic deafness are on the average more sensitive to emotional disturbances than other people it would seem that they may be hypersensitive not only after the deafness from otosclerosis supervenes but also long before its advent, and whether this hypersensitivity was inherited

or acquired. It is certain that emotional instabilities present before the onset of deafness will influence the reactions to the deafness, of the patient, his family and friends.

It is now believed that there are genetically controlled stimulii and reflexes which lead to basic instincts and behavior patterns. These patterns are in large degree inherited as potentialities. It follows that a child of a parent with a hypersensitive autonomic system will in a considerable number of cases inherit such potentialities or predispositions. In addition he will be subjected environmentally to the exaggerated emotional reactions of the parent. He will react similarly to the parent who has the more to do with his social development (usually the mother). Mothers are commonly more closely associated with their daughters' lives than with their sons'. (Here there are many implications.)

The age incidence of otosclerosis and its varying locations and periods of activity as revealed by clinical experience and in Guild's[2] histologic data indicates that the lesion develops only after birth and in an increasing incidence ratio varying with certain age decades; that the lesion is latent in the bone long before it is recognizable morphologically, and active long before it is recognizable clinically. Almost all of the cases reported as otosclerosis before the age of 10 are open to question, and the ones under eight are all very questionable. Also it suggests that if autonomic neural factors are in fact implicated in the etiology or activity of the lesions, the age incidences, distributions and periods of activity would be variously spaced just about as they are. No other etiologic factors so far suggested fit into the pattern as well as this.

Long before birth, although the neurovascular like the respiratory systems are present and ready to function, they are primarily completely under maternal influence until after birth. Immediately after birth and all during infancy and childhood the emotional factors develop with increasing powers to cause neurovascular effects in the tissues near to and following puberty.

Autonomic neural readjustments with emotional instabilities are prominent near puberty, menstruation, pregnancy and the menopause, periods in which otosclerosis deafness is prone to increase. They are also associated with endocrine variations, some of which, as I have pointed out, are known to be capable of affecting bone metabolism.[3] These factors intensify autonomic sensitiveness and are repeated or prolonged at these periods of life and during any severe emotional experiences at any time. What I am trying to say is that all this fits in perfectly with the known facts concerning age incidence and exacerbations of otosclerosis clinically and histologically observed.

Emotional sensitivity or instability may be due to the deafness, increased by it, or due to causes other than the deafness, whether present before or after deafness supervenes.

To learn whether a patient's nervous temperament differs from his or her siblings before the event of the deafness is often very difficult.

In these investigations it is pertinent to keep in mind that the teen-aged or adolescents who develop deafness from otosclerosis are in several respects in a different category from the child who is deafened from other causes:

1. He is seldom if ever deafened before his teens, and therefore
2. Not until after he has acquired a large vocabulary and all that this connotes.
3. His deafness does not actually come on suddenly, nor usually is it noted markedly in one ear alone.
4. There is seldom pain or any operative procedure associated with it, so that
5. He is deprived of the kind of concern and sympathy and understanding on the part of his parents and family that painful and operative illnesses engender.
6. Everyone is unaware of his deafness at first because it is slight and there is no apparent reason for any deafness.

7. On the other hand, unlike the deafmute or the severely deafened child, he is not subjected to overconcern and protection. He is not relieved of responsibility or from doing things for himself. On the contrary, he, and especially she, is subjected to increasing obligations and family duties.

8. As he grows older he is subjected to increasing irritations because he does not hear perfectly and therefore may appear stupid and inattentive. He becomes progressively frustrated, apathetic and retiring, with loss of attention in school and at home. He either assumes a "dead pan" expression, tends to withdraw from social contacts or manifests an exaggerated eagerness, or puts on an act or displays of temper. He may talk too much and monopolize the conversation so as to avoid having to listen to others. A common trait is to break into and interrupt conversations, without regard to the context of the subject under discussion; to keep talking largely to prevent trying to hear what others are saying, etc. Because he hears his own voice well, he may speak softly with the result that everyone tends to speak to him softly. All of these reactions really upset not only him but his family and friends and accentuate his and their emotional reactions.

I find that almost all persons having otosclerosis deafness reveal many characteristics showing that as a group they are emotionally hypersensitive, such as

1. Persistent search for a magical cure when they have been repeatedly informed by competent authorities that there is no cure.

2. Lack of adequate cooperation with their physician.

3. Failure to admit the need of lipreading or hearing aids, and often violent objections to same; a false sense of shame.

4. Willingness to submit to the fenestration operation even when told by several competent otologists that they are not favorable subjects for this procedure.

5. Exaggeration of slight improvement in hearing (even 5 db at one or two frequencies) following the fenestration operation.

6. Exaggerated depression following failure of operation.

7. Exaggeration of a tinnitus which you can show them is not really as loud as they would have you believe.

8. Flushing in one or both cheeks on slight provocation.

9. Sweaty or cold hands or feet.

10. Bringing with them a long list of written questions.

11. Failing to listen to what others are saying, even to the otologist whom they consult.

12. Paying more attention to what they themselves are saying or desire to say than to what their physician is saying or trying to find out.

13. Many of the other symptoms known to the neuropsychiatrist, or revealed by the newer mechanical and electronic techniques.

For many years I have been under the impression that in families with otosclerotic deafness the oldest or the elder of the siblings in each family were the ones most likely to be affected; also that in large families, and especially if there was a lapse of several years before the youngest was born, the youngest was frequently the one with otosclerosis. Being the oldest or the youngest usually entails a markedly different environment in the family and outside of the family, even in the uterus, than that encountered by the other siblings. In the case of females the oldest girl often takes on much of the duties of the mother, and all the social limitations which this entails, and the youngest girl, or boy, may be unduly pampered, overprotected and spoiled. Although responsibilities would generally be less for the younger child, this does not necessarily make for ideal emotional balance, and many times the youngest child is subjected to more disturbing emotional upsets than the older children. On the other hand, being the

oldest furnishes a longer time in which the child can be subjected to emotional strains, and more time for an otosclerosis to develop.

With these ideas in mind I tabulated my observations in 224 cases of otosclerosis, with the following results: (In all instances where the exact ages or sequences were not obtained, this is indicated by a question mark (?) or the case is excluded from the calculations respectively.)

In females when there were two siblings, 72 per cent were the elder and 28 per cent were the younger.

In males when there were two siblings, approximately 50 per cent were the elder and 50 per cent were the younger (too few cases).

FEMALES		MALES	
Age of onset before 20	7	Age of onset before 20	6
Age of onset after 20	13	Age of onset after 20	8
Age of onset ?	1		
	21		14

In females when there were three siblings, 60 per cent were the eldest and 40 per cent were the youngest.

In males when there were three siblings, approximately 75 per cent were the eldest and 25 per cent were the youngest (too few).

Including those in the middle the distribution was:

FEMALES		IN FEW MALES EXAMINED	
Oldest	39.3 per cent	Oldest	66.6 per cent
Middle	33.0 per cent	Middle	16.7 per cent
Youngest	27.7 per cent	Youngest	16.7 per cent
	100.0 per cent		100.0 per cent

FEMALES		MALES	
Age of onset before 20	14	Age of onset before 20	3
Age of onset after 20	9	Age of onset after 20	6
Age of onset (?)	1	Age of onset (?)	2
	24		11

In the two and three sibling groups the ratio of females to males examined was roughly 2:1. In the low brackets of sibling number my impressions appear to have been substantiated.

When there were four siblings the distributions were about equal for all age sequences in males and females, and the age of onset before 20 and after 20 was about the same percentage.

When there were five or more siblings the distributions were:

FEMALES		IN THE MALES ABOUT THE SAME BUT TOO FEW
Oldest	13 per cent	
Next to oldest	6 per cent	
Middle	26 per cent	
Next to youngest	18 per cent	
Youngest	37 per cent	
	100 per cent	

FEMALES		Per Cent	MALES		Per Cent
Age of onset before 20	11	17.7	Age of onset before 20	6	35
Age of onset after 20	47	75.9	Age of onset after 20	10	60
Age of onset ?	4	6.4	Age of onset ?	1	5
	62	100.0		17	100

Only 13 were over 40 years of age when I first examined them. The ratio of females to males examined in the four and five sibling groups was above 3:1.

Herein there was a large percentage of younger (37 per cent) and next to younger (18 per cent) total 55 per cent; and middle placed siblings with otosclerosis (26 per cent), total 81 per cent; and a small percentage of next to oldest and oldest (19 per cent).

That the incidence was higher in the oldest than in the youngest, or younger in the smaller families was expected, but that the reverse woud be found in the larger families was unexpected.

In the higher sibling brackets my impressions were wrong except as to otosclerosis being frequently encountered in the youngest siblings; but it was not only frequent, it was almost three times that in the oldest of these siblings. Had I examined more patients in smaller families than in large families this unbalanced impression would be explainable, but the fact is that the reverse was true. I examined more patients in the

larger families than in the smaller families. This at first thought seemed to mean that all of my work was wasted; however, I noticed that the eldest and the youngest taken together did constitute by far the greater number of siblings with otosclerosis in all the age sequence categories. I intend to examine a larger number of patients in the expectation that something more informative will be revealed, that the data will be more significant.

In order to use controls who also were subjected to the reactions caused by noninflammatory deafness I chose patients with nerve deafness. In these neural deafness patients in no category of sibling number was there a higher incidence in the younger than in the older siblings. This was to be expected because of the greater number of years lived by the older siblings; but it did not obtain in the large otosclerosis sibling groups, although it would be logical to expect the larger the number of siblings the older the eldest would be, and the longer time he would be exposed to the etiological factors.

One more observation: For the past three years I have examined the circulation in the arterioles and venules in the occular conjunctiva of all my patients and thus far without exception have noted a sludging of the blood, and certain other vascular phenomena in every patient with deafness from otosclerosis.

In two sets of identical twins, the one in each pair having typical otosclerotic deafness repeatedly revealed sludging of the blood phenomena, whereas the twin who had no deafness showed either no sludging of the blood, or distinctly less than the one with otosclerosis.

All these otosclerosis patients, although usually believing themselves in perfect health, were emotionally disturbed, hypersensitive people. This is one explanation for the sludging of the blood, because autonomic reactions often cause stasis in the organs and tissues of the body with sludging of the blood and related phenomena. The sedimentation rate, although it tends to correlate with sludging, was seldom increased in these

patients. There is much more to this but I shall not go into it at this time. It is relevant to recall that all the theories now seemingly with the most plausible deductions from the observed pathology (and all such have been derived only from pathology) have been based on local congestion, stasis, endocrine and metabolic disturbances, or anoxia in the bony capsule or the neural tissues of the ear. All such theories have been derived only from morphological studies of the lesion, the dead end of the search. They do not explain the *cause* of otosclerosis. Not only is all of the terminal pathology of otosclerosis explainable by the hypothesis I herein set forth, but also a fundamental factor in etiology.

I submit that the immediate underlying cause of otosclerosis and any auditory neural deafness incidental to it is causable by autonomic dysfunction, stasis, anoxia, etc., as outlined in this paper. Whether it is one or the other or both of these disorders depends upon the capillary beds affected. No other explanation seems to me probable.

If my thesis is tenable, what can we do about it? Although in none of the older theories was there any hope for prevention or for control, now at least there is a glimmer of hope because emotional and behavior traits and reactions are in man susceptible to modification. Even a shortening of emotional reactions will help to some extent; moreover, liability and assets in the emotional nature may be assessed even as early in life as the preschool years, and appropriate medicinal and neuropsychological measures taken to improve defects. Such measures are indicated not only to insure a happier social life, but also a life without many of the ills otherwise impending. When there is an inherited or potential tendency to it, one of these ills is otosclerosis. Will anyone avail himself of such preventive measures? I wonder!

The approach I have outlined is beginning to pay dividends in such psychosomatic diseases as duodenal ulcer, diabetes and asthma. Why should not the otologist try to control the effects of emotional factors in otosclerosis?

REFERENCES.

1. Fowler, E. P.: Sudden Deafness. The Laryngoscope, 60:7:680-695, 1950.

2. Guild, S. R.: Histologic Otosclerosis. *Ann. Otol., Rhinol. and Laryngol.*, 53:246, June, 1944.

3. Fowler, E. P.: Estrogens, Bone Metabolism and Otosclerosis. *Arch. Otolaryngol.*, 48:6:637-644, Dec., 1948.

140 East 54th Street.

PLASMOCYTOMA: A CASE WITH ORIGINAL LESION ON THE EPIGLOTTIS AND METASTASIS TO THE TIBIA.*†

JAMES B. COSTEN, M.D.,
St. Louis, Mo.

The observation of a second case of plasmocytoma, whose primary tumor occurred in the upper respiratory tract, warrants reporting, with follow-up information of the first case.[1] The tumor in the first case was attached to the right base of the tongue by a broad pedicle, and in the second, to the rim of the epiglottis, extending to the adjacent pharyngeal wall. Both are associated with myelomas in distant medullary bone; however, neither have shown Bence-Jones protein in the urine.

The first case, age 63, diagnosed plasmocytoma, April 27, 1946, is symptom-free, working as a filling station operator. No radiation therapy was used on his bone lesions, which were ovoid areas of rarefaction in the right and left ilium. Films of cranium, chest, long bones and pelvis, made Jan. 10, 1951, showed none of these areas of rarefaction.

These two examples of plasma-cell tumor showing soft tissue and osseous lesions simultaneously have one early metastasis of the plasmocytoma to adjacent lymph nodes, and in both the appearance of the lesions in the medullary bone. Tumor activity within the hematopoetic system occurs in both cases, but no secondary anemia has been found. In Case 1 an adjacent cervical lymph node contained tumor, and in Case 2 a similar node was free of tumor. These are good examples of the paradoxical nature of every set of facts regarding plasmocytoma and multiple myeloma. Stout and Kenney[2] question the use of the word metastasis of this tumor, stating:

*Read before the meeting of the Middle Section of the American Laryngological, Rhinological and Otological Society, Inc., Cleveland, Ohio, Jan. 15, 1951.

†From the Department of Otolaryngology, Washington University School of Medicine and the Oscar Johnson Institute.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Jan. 18, 1951.

"Indeed it is quite possible that all of these so-called metastases represent examples of multiple primary involvement, in which the nasal or oral involvement preceded the involvement of other sites, often by a long period of time. If the secondary involvement of bone marrow is an example of true blood-borne metastasis, it seems almost incredible that there should

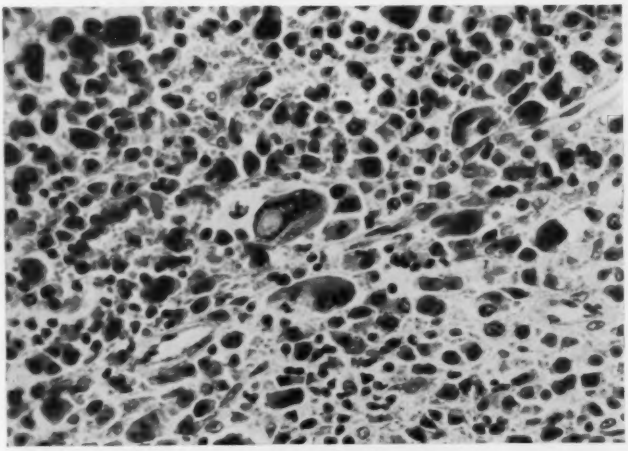


Fig. 1. ×600. Malignant plasma cell tumor, individual cells demonstrate pleomorphism with tumor giant cells, prominent nucleoli. A few typical plasma cells can still be recognized, although most are abnormal and many nuclei are disproportionately large in relation to cytoplasm. All plasma cell tumors with this microscopic pattern are malignant.—Lauren V. Ackerman, M.D., Surgical Pathologist, Washington University School of Medicine.

not have been at least some suggestion of lung involvement." Their report is a most complete review of 104 published cases of plasma-cell tumors of the upper air passages and oral cavity, adding seven new cases.

SUMMARY.

A case of plasmocytoma is herewith added to the record whose primary or principal growth was located on the epiglottis and pharyngeal wall and associated with a large focus of myeloma within the tibia. Increase of plasma cells in the

bone marrow and typical plasma cells in the tumor made the diagnosis, but the tumor shows atypical giant cells.

The responsibility of proper management rests on the laryngologist who sees the tumor first. It should be promptly removed and the area subjected to Roentgen therapy. The skeletal system is completely X-rayed to establish amount of dissemination.

The urine is checked for Bence-Jones protein, which, if present, indicates activity of tumor in the bone marrow.

Fig. 2. Anteroposterior and lateral views of the left knee demonstrate a large metastasis in the lateral condyle of the tibia: The metastasis is characterized by a large translucent area involving most of the lateral condyle of the tibia with extension into the metaphysis. This area is approximately 6 cm. by 7 cm. by 5 cm. The margins of the defect are well defined, but they are slightly irregular and rough, due to dissolution of the bone and its replacement by the tumor. Metastatic tumors occurring in bones distal to the knee and distal to the elbow are quite rare. This patient has not complained of pain in any other bones and a chest film made at this same date was negative for pulmonary metastases.—Wendell G. Scott, M.D., Associate Professor of Radiology, Washington University School of Medicine.

Serum proteins are checked, since high globulins are associated with plasmocytoma. Anemia is common as the patient enters the terminal phase. Repeated sternal punctures are

extremely important, increased plasma cells in the bone marrow showing presence of myeloma, thus altering the prognosis.

Broad field radiation is more desirable than interstitial elements. The case presented herewith is a good example of an isolated bone metastasis, apparently controlled by radiation, followed with curettement and repair of the bone defect with bone chips.

Case 2: G. P. R., white male, age 52, was admitted to McMillan Hospital on Sept. 1, 1949, for removal of a round mass attached to the left border of the epiglottis. It was 2 cm. in diameter and had the color and texture of lymphoid tissue. A small palpable soft gland was noted at the angle of the left jaw.

Surgery: On Sept. 2, 1949, the tumor was exposed under local anesthesia for the purpose of biopsy and removed with cautery. This proceeded with ease, and just as the spatula was removed from the base of the tongue a copious hemorrhage ensued. The exact area could not be found. Sponge pressure controlled it, but bleeding began again when taken away. At this point the left external carotid was tied, removing the enlarged lymph gland. Laboratory findings, Sept. 1, 1949, were not significant: Urine negative, no Bence-Jones protein; R. B. C. 4.79, W. B. C. 9,300, hemoglobin 98 per cent—15.2. Platelets normal; Schilling, bas. 2, eos. 2, stab. 1, seg. 74, lym. 15, mono. 6. The Kahn was negative.

Elaborate study was made of sections of the tumor, reported on Sept. 13, 1949. by Dr. Ackerman and Dr. Stephens as follows: "The surface exhibits stratified squamous epithelium which has been interrupted at one point, a fragment of cartilage opposite. At this point there is necrosis of the underlying tissue. The bulk of the tissue is composed of an extremely cellular mass which forms a rounded 'nodule.' This is composed of cells which appear generally in somewhat irregular sheets or strands and which for the most part have a moderately abundant cytoplasm and eccentrically placed hyperchromatic nuclei in which there is often some typical chromatin element. Throughout the section there are numerous giant cells; some of these having a pattern similar to those cells described above, while others appear multinucleated and less distinctive."

He was discharged from hospital Sept. 8, 1949, before the complete pathological studies were returned. Films of long bones, pelvis and chest were made on Sept. 12, 1949, by Dr. Wendell Scott, at which time radiation therapy was begun over the left cervical and jaw region and continued until Nov. 12, 1949, 38 exposures totaling 6,450 r.

Tenderness appeared over upper third of left tibia late in October, 1949; X-ray on Nov. 9, 1949, confirmed it as a metastasis (see Fig. 2). He entered Jefferson Barracks Hospital, Nov. 17, 1949, where a biopsy was made and this showed myeloma. He returned to Dr. Wendell Scott and between Dec. 12, 1949, and Jan. 7, 1950, received daily radiations over the upper third of the left tibia, totaling 3,750 r. The area remained painful and daily treatment was again given via another portal, from Jan. 9, 1950, to Jan. 14, 1950, for a total of 550 r.

Films of the area on Feb. 24, 1950, and March 24, 1950, showed the area of metastatic tumor unchanged, and he was admitted to Barnes Hospital

for study. Films of skull, chest, lumbosacral spine, cervical spine and pelvis were negative for metastasis. Repeated differential counts of the blood and sternal punctures on April 8, 1950, and April 11, 1950, showed normal erythroid and myeloid elements, but an increase in plasma cells, unevenly distributed in the marrow, some being young forms. This increase of plasma cells, together with the positive diagnosis of plasmocytoma in the original tumor, made a likely diagnosis of multiple myeloma possible.

Surgery: On April 15, 1950, the lateral aspect of the tibia was exposed by Dr. David Oliver, and a large cavity which represented the tumor was curetted, repacked with rib bone from the bone bank. Multiple sections of the tissue showed no definite evidence of persistence of tumor, indicating a high radiosensitivity of the neoplasm. It was the certain opinion of Dr. Ackerman that the tumor is present in other locations, and that the prognosis is hopeless. Palliative effort is advised when symptoms again appear.

He was discharged from Barnes Hospital, April 23, 1950, improved, with the left knee stiff but not painful. He remained the same when seen Dec. 14, 1950.

BIBLIOGRAPHY.

1. Costen, J. B.: Plasmocytoma: Report of a Case. *Trans. Amer. Laryngol., Rhinol. and Otol. Soc.*, pp. 432-438, 1947.

2. Stout, A. P., and Kenney, F. R.: Primary Plasma-Cell Tumors of the Upper Air Passages and Oral Cavity. *Cancer*, 2:261-278, 1949.

A GIANT RHINOLITH.*

CAESAR N. ABU-JAUDEH, M.D.,
Beirut, Lebanese Republic.

Although over 300 cases of rhinolith have been recorded,[1] the occurrence of enormous nasal concretions is comparatively rare. Melay,[2] Myerson[3] and Gorman[4] extracted nasal calculi that weighed 85 gm., 56 gm. and 70 gm., respectively. The largest calculus recorded in the literature weighed 110 gm. and required Rouge's sublabial operation for its removal.[5]

The aim of this paper is to describe a nasal calculus which weighed 116 gm. (6 gm. heavier than the largest recorded) and which had greatly distorted the facial bones, causing symptoms suggesting a malignant growth.

REPORT OF A CASE.

S. H., a 50-year-old Arab male, was admitted to the ear, nose and throat service on Nov. 20, 1950, complaining of bilateral nasal blockage, excessive tearing from the right eye and foul discharge from the right nostril for the past 10 years. During the last two years he had noticed a gradual displacement of his right eye, a fullness of his right cheek and widening of the bridge of his nose. He did not complain of pain or epistaxis.

Examination revealed a marked outward and downward displacement of the right eyeball and an evident swelling of the right side of the nose and adjacent cheek region. A stony hard mass could be felt springing from the medial wall of the right orbit. The right nasal cavity was occupied by a large stony hard mass. A thick grayish discharge came from around the mass and had a foul, pestilential odor. The left nostril was obstructed by a displacement of the septum to that side. Posterior rhinoscopy showed a grayish mass occupying the right side of the nasopharynx and extending to the left, leaving a small slit-like opening in place of the left choana. There was no sign of the posterior edge of the nasal septum. Both nasal bones and also the nasal process of the left maxilla were displaced outward and to the left. X-ray study by Dr. G. Saleeby revealed a large irregular body occupying the nasal cavity

*From the Department of Otolaryngology, American University of Beirut, Lebanese Republic.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, Feb. 1, 1951.

and encroaching on the right maxillary and ethmoidal sinuses and on the right orbit. It was either a rhinolith or a bony mass; more probably the former.

Attempts to break up the mass or take a biopsy were unsuccessful on account of its hard consistency. An external operation to deliver it was, therefore, found necessary.

The operation was performed under pentothal, nitrous oxide and oxygen intratracheal anesthesia. An incision was made from the inner ex-

Fig. 1. Roentgen film of lateral view of nose showing rhinolith in situ.

tremity of the right eyebrow descending along the nasobuccal groove to the side of the ala nasae. The bone was exposed and divided according to Moure's lateral rhinotomy technique, thus obtaining a large opening through which the mass was grasped and, after considerable difficulty,

dislodged and extracted. The mass was ivory-hard, grayish black, and presented a comparatively smooth, rounded lateral surface which fitted snugly into the right maxillary and ethmoidal sinuses, and a medial (septal) rough surface which had fractured and pushed the septum

Fig. 2. Roentgen film of anteroposterior view of skull before operation. Note encroachment of rhinolith on right maxillary and ethmoidal sinuses and its extension into the right orbital cavity.

against the lateral wall of the left nostril. It measured 7 cm. by 6.2 cm. in its greatest diameters and weighed 116 gm. (see Figs. 1 and 2).

Inspection of the nasal cavity now revealed complete absence of the lateral wall of the right nostril (the turbinates, the nasal surface of the maxilla, the ethmoidal labyrinth, the orbital plate of the ethmoid bone and the lacrimal bone). Only part of the anterior cartilaginous septum had remained and this was replaced centrally by a septum forceps. Like-

Fig. 3. Rhinolith, lateral view.

Fig. 4. Rhinolith, front view.

wise, both nasal bones were fractured and pushed back in place. The nose was cleansed as much as possible and packed with iodoform gauze to control bleeding. The external wound was closed with cotton sutures.

Two days later the packing was removed. Penicillin therapy was started on the day of operation and continued for nine days. The postoperative course was uneventful and on Dec. 6, 1950, 12 days after operation, the patient was discharged as recovered.

Fig. 5. Roentgen film taken 10 days after removal of the rhinolith. Lateral view.

Chemical analysis (No. S-381-50) of the rhinolith by Prof. W. A. West revealed the following: Organic matter plus moisture, 33 per cent; calcium phosphate, 43.7 per cent; calcium carbonate, 21.8 per cent; undetermined, 1.5 per cent.

SUMMARY.

1. Large rhinoliths are rare.

2. A rhinolith weighing 116 gm. (6 gm. heavier than the largest recorded) and simulating a malignant growth of the nose is reported.

3. A radical external operation was required to remove it.

Fig. 6. After removal of rhinolith, anteroposterior view.

4. Photographs of the rhinolith and also preoperative and postoperative Roentgen films of the patient are presented.

BIBLIOGRAPHY.

1. Thomson, Sir St. Clair, and Negus, V. E.: Diseases of the Nose and Throat; a Textbook for Students and Practitioners. 5th Ed. New York, N. Y.: Appleton-Century-Grofts, Inc., pp. 199.

2. Melay, A. B.: *Ann. d. mal. de l'oreille, du larynx*, 32:91, Jan., 1906.

3. Myerson, M. C.: The Laryngoscope, 38:393, 1928.

4. Gorman, J. R.: *Trans. Am. Laryngol., Rhinol. and Otol. Soc.*, 36:498, 1930.

5. Motey, R.: *Ann. d. mal. de l'oreille, du larynx.*, 32:496, Nov., 1906.

EYE, EAR, NOSE AND THROAT HOSPITAL ALUMNI ASSOCIATION.

The first annual meeting of the Eye, Ear, Nose and Throat Hospital Alumni Association will be held in New Orleans, La., April 26, 27 and 28, 1951. All physicians who have received their specialty training at the hospital are cordially invited to attend.

One day will be devoted to ophthalmology and another to otolaryngology. On the morning of Saturday, April 28, 1951, there will be demonstrations of a number of operative procedures in each specialty.

Each night there will be a cocktail party. During the day the wives of the alumni will be entertained at tea, taken on tours of homes in New Orleans' famed French Quarter and Garden District, and taken on a yacht trip on the Mississippi River, during which a buffet luncheon will be served.

Please address all inquiries to Geo. H. Hauser, M.D., 145 Elk Place, New Orleans, La.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The 1951-1952 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on Sept. 1 and continue for a period of 10 months. Registrations must be completed before Aug. 15. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minn.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

March 1, 1951.

Aurex Model F and Model H.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Harmony Mono-Pac; Beltone Symphonette; Beltone Mono-Pac Model M.
Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dysonic Model 1.
Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.
Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Maico Atomeer; Maico UE-Atomeer; Maico Quiet Ear Models G and H.
Manufacturer: Maico Co., Inc., North Third St., Minneapolis, Minn.

Mears Aurophone Model 200; 1947—Mears Aurophone Model 98.
Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 101 (Magnetic Receiver); Micronic Model 303. (See Silver Micronic.)
Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T-3 Audiomatic; Microtone T-5 Audiomatic; Microtone Classic Model T9; Microtone Model 45.
Manufacturer: Microtone Co., 4602 Nicollet Ave., Minneapolis 9, Minn.

National Cub Model C; National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.
Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-1S; Otarion Model E-2; Otarion Model E-4; Otarion Models F-1, F-2 and F-3.
Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Models VH and VL (Standard); Paravox Model XT (Xtra-Thin); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).
Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet.
Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Silver Micronic; Silver Micronic (Magnetic and Crystal) Models 202M and 202C. (See Micronic.)
Manufacturer: Micronic Corp., 101 Tremont St., Boston 8, Mass.
(See Micronic.)

Silvertone Model 103BM.
Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.
Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model M-35.
Manufacturer: Microme Co., 727 Atlantic Ave., Boston 11, Mass.
Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Silvertone Model P-15.
Manufacturer: W. E. Johnston Mfg. Co., 708W. 40th St., Minneapolis, Minn.
Distributor: Sears-Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.
Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 600; Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.
Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago, Ill.

Televox Model E.
Manufacturer: Televox Mfg. Co., 117 S. Broad St., Philadelphia 7, Pa.

Telex Model 22; Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 1700.
Manufacturer: Telex, Inc., Minneapolis 1, Minn.

Tonemaster Model Royal.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Trimm Vacuum Tube No. 300.
Manufacturer: Trimm, Inc., 400 W. Lake St., Libertyville, Ill.

Unex Model "A"; Unex Midget Model 95; Unex Midget Model 110.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Model J.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Model 63; Western Electric Model 64; Western Electric Models 65 and 66.
Manufacturer: Western Electric Co., Inc., 120 Broadway, New York 5, N. Y.

Zenith Model 75; Zenith Miniature 75.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago (10), Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Kenneth M. Day, 121 University Place, Pittsburgh, Pa.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Meeting: Hotel Claridge, Atlantic City, N. J., May 11-12, 1951.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Meeting: Hotel Claridge, Atlantic City, N. J., May 9-10, 1951.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
Secretary: Dr. C. Stewart Nash, 108 Medical Arts Bldg., Rochester, N. Y.
Meeting: Hotel Claridge, Atlantic City, N. J., May 6-8, 1951.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. James M. Robb, 641 David Whitney Bldg., Detroit, Mich
Vice-Chairman: Dr. J. M. Robison, 1304 Walker Ave., Houston 2, Tex.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3, Tenn.

AMERICAN BOARD OF OTOLARYNGOLOGY.

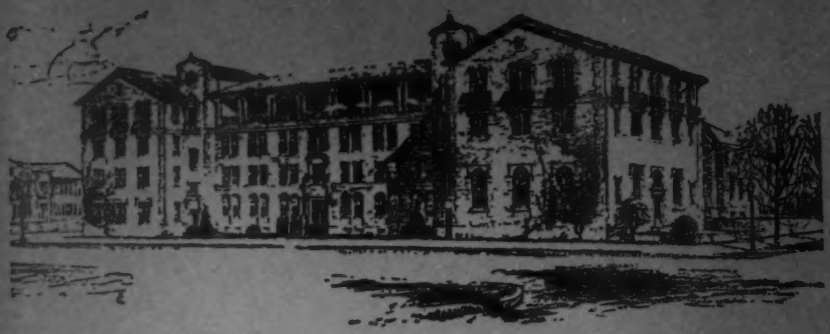
Meeting: Hotel John Marshall, Richmond, Va., May 1-4, 1951.
Palmer House, Chicago, Ill., Oct. 9-12, 1951.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. Victor Alfaro.
Vice-Chairman: Dr. Irvin Feldman.
Secretary: Dr. Frasier Williams.
Treasurer: Dr. John Louzan.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. J. Mackenzie Brown, 1136 W. 6th St., Los Angeles, Calif.
President-Elect: Dr. Derrick Vail, Chicago, Ill.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.



Central Institute for the Deaf

NATIONAL RESIDENTIAL AND DAY SCHOOL FOR THE DEAF AND DEFECTIVES IN SPEECH

Approved by Advisory Council of Foremost Ear Specialists and Educators

New fire-proof buildings beautifully located opposite Forest Park. Modern Dormitories and Equipment. Best home environments. Pupils constantly in care of teachers or experienced supervisors.

ORAL SCHOOL FOR DEAF CHILDREN

C. I. D. offers all advantages of exclusively Speech Training and expert medical supervision for both Resident and Day Pupils.
Nursery School (2 years of age) through the Elementary Grades.

ACOUSTIC TRAINING FOR CHILDREN WITH RESIDUAL HEARING

Salvaging of Residual Hearing is a specialty of C. I. D. The Acoustic Method was created here. Group and individual hearing aids used for class instruction at all grade levels.

LIP-READING INSTRUCTION

Private and Class Instruction for Hard-of-Hearing Adults and Children.
Conversational Classes for advanced pupils. Speech conservation stressed.

CORRECTION OF SPEECH DEFECTS

Private and Class Instruction for children with normal hearing and delayed speech or defective speech.
Resident and Day Pupils (2 years of age through Elementary Grades).
Private Instruction for Adults.
Correction of Imperfect Phonation, Imperfect Articulation, Aphasia, Stuttering.

TEACHERS TRAINING COLLEGE

Two years of Training following a professional curriculum for applicants with adequate college qualifications. Graduates qualify for degrees of Bachelor of Science in Education or Master of Science in Education from Washington University. Graduates prepared to teach both the deaf and speech defective.

DR. MAX A. GOLDSTEIN, Founder DR. HELEN SCHICK LANE, Principal

818 S. KINGSHIGHWAY 10, ST. LOUIS, MO.

CONTENTS

NASOPHARYNGEAL CANCER. Daniel Miller, M.D., Boston, Mass. - 187

THE PERMANENTLY PATENT FENESTRA NOV-OVALIS. Julius Lempert, M.D., New York, N. Y. - - - - - - - - - - - - 215

HISTOPLASMOSIS IN THE ADULT: CASE REPORT AND REVIEW OF THE LITERATURE. Dean M. Lierle, M.D., and T. Lyle Carr, M.D., Iowa City, Iowa - 230

BRONCHIAL ADENOMA. Chevalier L. Jackson, M.D., and Charles M. Norris, M.D., Philadelphia, Pa. - - - - - - - - - - 238

ANTRO-ALVEOLAR FISTULAE. Frederick T. Hill, M.D., Waterville, Me. 242

EMOTIONAL FACTORS IN OTOSCLEROSIS. Edmund Prince Fowler, M.D., New York, N. Y. - - - - - - - - - - - - - 254

PLASMOCYTOMA: A CASE WITH ORIGINAL LESION ON THE EPIGLOTTIS AND METASTASIS TO THE TIBIA. James B. Costen, M.D., St. Louis, Mo. - - - - - - - - - - - - - - - - - 266

A GIANT RHINOLITH. Caesar N. Abu-Jaudeh, M.D., Beirut, Lebanese Republic - - - - - - - - - - - - - - - - 271

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION - - - - - - - 279

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES - - - - - - - 282